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C. Everett Koop, M.D.
Consulting Editor

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Forthcoming Symposia:

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FOREWORD

C. EVERETT KOOP, M.D.

Consulting Editor

FABIAN BACHRACH

The pediatrician has been so successful in the management of most of the illnesses presented by children that many of the admission diagnoses of even a decade ago are uncommon on pediatric services today. Because of this the surgical lesions of children have become relatively more frequent. In addition, advances in the understanding of neonatal physiology as well as of newer surgical techniques and specific diagnostic procedures have produced an absolute increase as well. In many children's hospitals about the country, surgical diagnoses account for approximately 70 per cent of admissions. Obviously, then, this branch of pediatric care has become an extremely important one to the medical pediatrician and the general practitioner.

When the publishers of *Pediatric Clinics* invited me to be consulting editor for a surgical number, it seemed an appropriate time to ask some of the pediatric surgeons in the United States and Canada to prepare a series of clinics directed toward the medical pediatrician and general practitioner, but with the surgical insight gained by long association with the unique problems of children.

Pediatric surgery has not been established as a specialty on this continent as long as it has been in Europe. In general it is looked upon with approval by pediatricians, but still being somewhat embryonic

in development, it is regarded with a certain amount of suspicion by general surgeons. Their concern, an understandable one, is that the field of surgery not be further splintered by another specialty. Most surgeons who devote all their endeavors to children agree wholeheartedly with this point of view, and indeed if ever there was a general surgeon today, it is a pediatric surgeon. This specialist considers the anatomic, physiologic, metabolic and psychologic needs of his diminutive patient to be a more natural area of horizontal specialization than the vertical separation according to anatomic organs and systems.

The contributors to this issue do not suggest that all children's surgery be done by children's surgeons, but rather acknowledge the fact that with infants and young children especially, mortality and morbidity seem to drop when pediatric surgical patients become the sole concern of a surgeon instead of part of a much broader field of surgical endeavor.

In general the orientation of each clinic is toward the pediatrician, but obviously some surgery has been included in order to substantiate diagnostic procedures and because postoperative care of many pediatric surgical patients is currently handled by the medical pediatrician in contradistinction to the usual postoperative management of the adult surgical patient.

Obviously, there is frequently more than one way to manage adequately a surgical problem. Some of the authors present methods of diagnosis and management quite different from those I use. This does not mean that my way is right and theirs wrong, nor is the converse true. Rather, it means that there is more than one good method of management, and the point of view expressed represents experience with a technique or procedure which the author has found to be satisfactory in his hands, and perhaps superior to others he has tried.

Subject matter in several of the clinics may seem to overlap. A lesion such as atresia of the esophagus, for example, may be covered by the physician interested in early diagnosis of surgical anomalies incompatible with life; it may also be covered by another concerned with respiratory obstruction, or it may be dealt with in the clinic concerned with proper timing of pediatric surgical procedures. This overlapping helps to emphasize the many facets in the management of pediatric surgical patients, and the several points of view can do nothing but add to a well rounded understanding of at least part of the problem.

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FACTORS AFFECTING SUCCESS AND FAILURE IN NEONATAL SURGERY

HARRY C. BISHOP, M.D.

Not all infants born with major congenital defects can be salvaged, but experience has shown that the salvage rate improves in medical centers which have devoted special attention to these problems. These congenital anomalies incompatible with life, but amenable to surgical correction, fall into five main groups: (1) esophageal atresia; (2) intestinal obstruction—(*a*) atresia, (*b*) malrotation with volvulus, (*c*) congenital bands with obstruction, (*d*) meconium ileus; (3) imperforate anus; (4) diaphragmatic hernia; (5) omphalocele.

A consideration of the factors influencing the mortality in these neonatal emergencies should be helpful, particularly to physicians who in their own institutions have not experienced an improvement in their neonatal mortality statistics.

UNCONTROLLABLE FACTORS AFFECTING MORTALITY

Prematurity

Unfortunately, serious congenital anomalies and prematurity are commonly associated. It has been estimated that approximately 25 per cent of the cases of esophageal atresia occur in infants born weighing under 5 pounds and, hence, considered premature by the usual standards.[2] Although many premature infants have had successful surgical procedures, the survival rate cannot be expected to equal that obtained with the larger, full-term infants.

Gross[3] has reviewed his experience with the premature infant requiring surgery and offers many helpful suggestions for his specialized care.

From the Surgical Clinic of The Children's Hospital of Philadelphia and the Harrison Department of Surgical Research, University of Pennsylvania School of Medicine.

Multiple Anomalies

Frequently an infant may have more than one congenital anomaly. This is not surprising, since, during the early weeks of gestation, many organs are simultaneously undergoing their early embryologic development at a time when the unknown insult occurs. The presence of multiple anomalies may not be immediately apparent, and therefore the physician must be alert to the possibility of and should actually search for other unsuspected anomalies that may complicate the newborn's convalescence.

Certain anomalies seem to occur concomitantly, and the possibility of such an association must be kept in mind. There is a higher incidence of esophageal atresia occurring with atresias lower in the intestinal tract or with an imperforate anus than is found with other congenital defects. Esophageal atresia is not uncommonly associated with congenital heart disease. The more severe forms of congenital heart disease can be detected early in infancy, although the exact anatomic defect of the heart usually defies accurate classification. Many of the less serious congenital heart defects are not even suspected during the neonatal period, and prolonged follow-up of any infant with one known congenital anomaly is essential.

Imperforate anus is frequently associated with urinary tract anomalies which may, of themselves, not be emergencies, but should be recognized so that urinary antibiotics can be given to prevent a complicating urinary tract infection. Infants who have had an anoplasty for the correction of imperforate anus should have an intravenous pyelogram as a survey of the urinary tract unless carried out preoperatively.

Anatomic Variations of Congenital Defects

It is important to recognize that the anomalies under discussion have many anatomic variations. For example, an atresia of the esophagus, with the usual blind upper end of the esophagus, may or may not have an assocated tracheo-esophageal fistula to the lower esophageal segment through which air enters the intestinal tract below. If there is no air in the stomach, it can probably be predicted that there will be an absent lower esophagus or a considerable distance between the upper and lower ends of the esophagus, so that primary anastomosis will be difficult or impossible (see also pp. 958, 1029).

The most common diaphragmatic hernia is a defect in the posterolateral aspect of the diaphragm through which abdominal viscera herniate up into the left pleural cavity. There usually is a rim of muscle posterolateral to the defect in the diaphragm, but this may be absent.

making the repair considerably more difficult and perhaps necessitating a type of thoracoplasty. Generally there is no covering over the herniated viscera, but a thin, transparent sac, attached to the rim of the diaphragmatic defect, can be present. This sac, with its contained viscera, can produce the same compression of the lung and leads to a dangerous shift of the mediastinum, as in the sacless variety (see also pp. 1038, 1155).

Only by familiarity with the anatomic variations that do occur, can the surgeon promptly recognize the problem presented and choose the operative procedure most likely to succeed.

CONTROLLABLE FACTORS AFFECTING MORTALITY

Promptness of Operation

With all neonatal anomalies it is axiomatic that the sooner the operation is performed, the better! The infant with esophageal atresia will have progressive pneumonia from the aspiration of secretions which pool in the blind upper esophageal pouch. Reflux of gastric contents through the fistula between the lower segment of the esophagus and the trachea will also produce a severe pneumonia during any period of delay.—The newborn with a diaphragmatic hernia is generally free of serious respiratory embarrassment at, and for a variable period after, birth. Only as the intestinal tract fills with swallowed air do the various intestinal loops in the pleural cavity expand, producing a further mediastinal shift and compressive atelectasis of the ipsolateral as well as the contralateral lung.—Most cases of intestinal obstruction have an undistended abdomen at birth, and only as air is swallowed do the intestines proximal to the obstruction become distended, producing an increase in intra-abdominal pressure which impairs the normal excursion of the diaphragm. Respiratory exchange is thereby limited, dyspnea ensues, and the infant rapidly becomes exhausted and, hence, a poorer candidate for operation.

Preoperative Study and Care

Early Diagnosis. An omphalocele and an imperforate anus should be diagnosed by inspection at birth. Other congenital anomalies are diagnosed early only by observation and investigation of abnormal signs occurring in the newborn infant.—Some cases of diaphragmatic hernia can be diagnosed shortly after birth by noting an asymmetrical expansion of the chest, an increase of the anteroposterior diameter of the chest and the presence of a scaphoid abdomen.—Any infant who shows difficulty in handling his pharyngeal mucus can easily have a soft, red, no. 8 rubber catheter passed through the esophagus to prove

its patency.—An infant who vomits bile-stained material must immediately be suspected of having a mechanical intestinal obstruction below the ampulla of Vater, and survey films of the abdomen should be taken promptly.

Delay in diagnosing these congenital defects implies either professional ignorance or a poorly managed neonatal nursery. The physician who accepts the care of newborn infants should be a policeman, accustomed to watching normal activity, but immediately suspicious of any alterations in behavior which could mean trouble ahead.

Minimal Diagnostic Procedures. The newborn infant requiring operation is facing the most dangerous experience of his life. His tolerance is surprisingly good for corrective surgery, provided he is not weakened either from delay, and its resultant complications, or from iatrogenic causes. For example, if a 10-hour-old infant has vomited bile-stained material, probably all that is needed are supine and erect roentgen films of his abdomen to show dilation of the upper intestinal tract proximal to an area of complete obstruction. A competent diagnostician can recognize the dilatation with air in the proximal loops of bowel and the absence of gas in the distal bowel below the level of the mechanical obstruction. The infant does not need further prolonged study in a darkened fluoroscopic room using barium from the stomach downward, or barium in a small, as yet unused, colon inserted from below. He does not need extensive blood chemical determinations. He does not need a further period of observation to substantiate the diagnosis. The diagnosis of mechanical obstruction is sufficient to warrant a laparotomy, and time should not be spent making extraordinary efforts to pinpoint the exact area or the cause of his obstruction.

The infant with abnormal bubbles of gas in the left pleural cavity, a shift of the mediastinum to the right and a diminished abdominal gas pattern does not need a barium meal to make the diagnosis of diaphragmatic hernia and to indicate the need for immediate operation.

The Use of Antibiotics and Vitamin K. All newborns undergoing surgery should be given antibiotics. These should be given as soon as possible in order to have adequate blood levels at the time of operation. Aqueous penicillin and streptomycin given intramuscularly are satisfactory. Vitamin K in small dosage is also indicated to prevent hypoprothrombinemia, which may increase the bleeding at the time of operation or cause complications during the postoperative period.

Intestinal Decompression. Once a correctible anomaly is suspected, further distention of the intestinal tract should be prevented. The infant with esophageal atresia should have a catheter with a single hole at its tip placed in the upper blind esophageal pouch. This can be intermittently aspirated to prevent an overflow and aspiration of secretions into the trachea. All other infants should have a multiholed

no. 8 red rubber urethral catheter passed through the nose, down into the stomach, and constant suction applied. The newborn infant with a mechanical obstruction of the small bowel has a relaxed pylorus, and gastric suction alone removes not only swallowed air, but also secretions passing from small bowel back into the stomach. It is desirable to prevent swallowed air from passing into the intestines not only when there is an obstruction, but also when the bowel is in an abnormal position as in a diaphragmatic hernia or an omphalocele. The repair of these defects will be easier if the intestinal loops are empty of gas so that less space is occupied by them when they are replaced in the abdomen.

There is seldom need for a long tube for intestinal decompression. The small caliber of the neonatal bowel and the need for immediate operation generally preclude the use of a tube which passes through the pylorus and down into the small bowel.

Safe Transportation. Safe interhospital transportation of an infant born with a congenital anomaly which requires emergency lifesaving surgery is not only feasible, but also frequently advisable. Specific suggestions for the safe conduct of the transportation have been outlined,[1] and the need for such transfer should not keep the newborn infant from having the best surgical and postoperative care in another institution which is equipped and staffed for this care.

The Operation

Intravenous Cut-Down for Maintenance of Blood Volume. All newborn infants, without exception, should have a small plastic catheter placed in either an ankle or arm vein prior to the start of any corrective surgical procedure. The cut-down can be done quickly even in the smallest infant, and its presence assures a reliable route for replacement of the blood lost. Single-donor fresh frozen plasma can be used for the first 25 or 50 ml. of blood lost if the infant has an initially high hemoglobin. Type-specific, compatibly cross-matched blood should be given to replace greater losses. The operator must learn to judge blood loss accurately and replace it as it is lost rather than waiting for the possibly irreversible signs of a depleted blood volume. A blood loss of 50 ml. in a 6½-pound infant represents 20 per cent of his blood volume, which would be equal to 1300 ml. in a 150-pound adult.

Anesthesia. Undoubtedly, the management of the anesthesia (see also p. 1003) given to any newborn may spell failure or success. We believe that all these congenital defects requiring emergency surgery are best repaired under inhalation anesthesia. Adequate relaxation of general anesthesia allows a faster, smoother and probably a safer operation when compared with the inherent difficulties of operating under

local, infiltrative anesthesia. To use general anesthesia safely, it is essential to have an anesthesia department particularly interested and experienced in the special problems of these small patients.

In our institution all newborns are operated upon with endotracheal anesthesia, which gives complete control of the infant's respiratory exchange and level of anesthesia at all times. The possibility of aspiration during manipulation of the stomach and intestines is lessened, and it is possible to use a muscle relaxant which greatly aids the repair of diaphragmatic hernias and omphaloceles and the closure of abdominal laparotomy incisions which might otherwise be difficult. All infants have a constant recording of their rectal temperature, and their cardiovascular status is monitored with an electrocardiogram tracing on an oscilloscope.

With proper care, in the hands of a skilled anesthesiologist, there should be a very low incidence of morbidity due to the endotracheal intubation and the management of the anesthesia generally.[4]

Techniques, Instruments, Sutures and Dressings. A great deal could be said about the surgical procedures themselves. Certainly some specialized techniques are required when dealing with these congenital defects, but by and large the directness of the operative approach, the gentleness in handling the tissues, and the fineness of the technique are the most important factors. A concerted effort should be made to do the definitive corrective procedure at the time of the original operation. Infants tolerate one operation of moderate length very well if it fully corrects the defects present. Repeated anesthesias without adequate intervals between them and partially corrective surgical procedures may be doomed to failure.

Transverse abdominal incisions are satisfactory for laparotomies in a newborn. A midabdominal transverse incision allows good exposure for all areas within the abdominal cavity. If necessary, the incision can be extended across the midline with safety.

Fine suture material, either absorbable or nonabsorbable, should be used. Careful approximation of all abdominal layers with multiple interrupted sutures is essential and should practically eliminate evisceration as a postoperative complication.

Abdominal skin incisions can be satisfactorily closed with buried subcuticular sutures of fine (5–0) nonabsorbable silk sutures. The wound can then be covered with water-tight collodion, which prevents the necessity of a thick gauze dressing held by adhesive plaster. This is a particularly useful method of closing lower abdominal incisions, where it is inevitable that contamination from stool and urine will occur.

Thoracic incisions are generally closed with a regular running suture or interrupted skin sutures of silk. It must be remembered that even

a single strip of adhesive plaster can limit the normal excursion of a newborn's thoracic cage. These dressings should be minimal in size, loosely applied, and removed at the earliest possible date.

Postoperative Management

FREQUENT OBSERVATION OF THE PATIENT. Newborn infants must be watched closely during the postoperative period. The infant's status can drastically change in a short time. Nursing care should therefore be continuous, preferably by a nursing team particularly trained in the care and postoperative complications that occur in the newborn. An experienced nurse is capable of determining at a glance significant changes in respiration that might require pharyngeal aspiration, aspiration of a chest catheter or cleansing of a nasogastric catheter. Prompt action initiated by such observations can be lifesaving.

Observation by a physician must be frequent during the postoperative period. It will not suffice to see the infant only once or possibly twice each day. These infants must be checked by the attending surgeon or a member of his resident staff at intervals not to exceed three or four hours, and many an infant has been salvaged because the surgeon remained with him constantly during the immediate postoperative hours.

CONTROL OF THE INFANT'S ENVIRONMENT. A modern incubator is essential for the postoperative care of the neonate. We have found the Isolette* an excellent incubator, for not only does it isolate, thereby lessening the incidence of cross-infections, but also its plexiglass construction allows constant observation of the infant without disturbing him. This modern Isolette allows regulation of the heat, oxygen supply and humidity, all of which are vitally important to a successful convalescence.

Many infants have an immature heat-regulating mechanism. By automatic controls the temperature within the Isolette can be regulated to maintain the infant's temperature in a satisfactory range. Extra oxygen is ordinarily supplied during the early postoperative period. Its concentration, however, is carefully controlled in order to lessen the danger of retrolental fibroplasia, which is a particular danger in the premature infant. Extra humidity can be maintained by bubbling either oxygen or air through sterile water. A humid environment lessens the tenacity of pharyngeal and tracheal secretions so that they can be handled more easily by the infant. If extra humidity is not supplied, small plugs of inspissated mucus may lead to a chronic and, perhaps, unrecognized hypoxia.

* Air-Shields, Inc., Hatboro, Pa.

Fear of Aspiration. One of the greatest postoperative dangers to a newborn is vomiting associated with a depressed cough reflex, which can result in aspiration and insurmountable pneumonia. This hazard can be lessened only by careful attention to gastric toilet by using either a nasogastric tube or a prophylactic gastrostomy for gastric decompression. If a nasogastric tube is used, it must have extra holes cut near its tip, and it must be irrigated, preferably with a sterile saline solution, at intervals to assure adequate function.

It is dangerous to write specific orders for oral feedings until you are sure that the infant's stomach can empty in a normal fashion. When feedings are started, it is safest to feed by gavage. First a known amount is instilled into the stomach. In two or three hours the stomach is aspirated to prove adequate emptying before the next gavage feeding is instilled.

Not only can feedings be aspirated, but also the usual secretions in the oropharynx are often poorly handled by a weakened infant. The pharynx in such an infant should be aspirated as needed, but no more than really is necessary. Edema and an increase in the outflow of secretions result if the mucous membranes are irritated too often by an aspirating catheter. If, unfortunately, the infant does aspirate either pharyngeal secretions or gastric content, this should be immediately recognized. A laryngoscope, which should always be immediately available, can expose the larynx, and the trachea can be gently cleared by aspirating with a small plastic catheter. This, too, can be a lifesaving procedure, and anyone caring for these infants should be skilled in its use.

Use of Antibiotics. Antibiotics should be given to all newborns during the postoperative period. These infants can develop septicemia not only from contamination at the site of operation, but also from persistent atelectasis, aspiration pneumonia, and from infections around the stump of the umbilical cord. We usually give penicillin and streptomycin intramuscularly rather than using the broad-spectrum antibiotics, which seem to lead to a higher incidence of thrush and diarrhea.

Intravenous Fluids and Electrolytes. Usually the intravenous cutdown placed at the time of operation can be effectively used for parenteral alimentation during the first two or three postoperative days. If intravenous infusions are needed after this time, they can be given into a scalp vein through a small short-beveled needle.

It has been learned that surgical infants do better "slightly on the dry side." It must be remembered that the newborn infant has immature renal function[6] and cannot easily handle overloading of fluid given intravenously. Only the necessary amounts of water and electrolytes are given to replace the urine, intestinal and insensible water

losses.[5] Salt-poor human albumin, plasma and blood should be used if parenteral alimentation is necessary for more than three or four days or as indicated by blood or protein losses.

Newborn infants have been successfully carried on intravenous therapy for a month or more, *but even with the newer intravenous solutions, adequate replacement of all the necessary elements intravenously is impossible.* Obviously, it is desirable to switch to oral alimentation as soon as possible. However, one must be sure the intestinal tract is capable of taking care of the quantity and type of nutrient offered before intravenous fluids are stopped.

Minimal Procedures. All infants during their convalescence require a "hands-off policy" except when essential treatments are being carried out. There is a great tendency to disturb and handle these patients too frequently rather than allowing them the necessary peace and rest required. The overzealous nurse who is constantly changing the baby's position or unnecessarily aspirating his pharynx or taking his rectal temperature can, in the critically ill infant, cause undue fatigue and lead to his demise. It takes considerable experience to know how much and how little need be done for these small patients.

Most of these patients can be cared for without frequent blood cell counts, urinalyses, and "across the board" blood chemistry determinations. Efforts should be made to sharpen clinical acumen to the point of knowing the infant's requirements from experience, and minimizing the need for extra laboratory help.

UNKNOWN FACTORS AFFECTING MORTALITY

Little is known about the stress of operation upon newborn infants. What metabolic changes occur due to pituitary-adrenal response? What alterations occur in liver and kidney function? What causes the increase in hemolysis of blood during the postoperative period?

Research studies are difficult to carry out in an ill, 6-pound infant, but it can be hoped that additional worth-while data will accumulate so that the explanation of "poor protoplasm" will no longer be used when discussing postoperative infant mortality.

PERSONNEL—"THE TEAM"

In conclusion, it can be stated that successful care of the newborn infant undergoing corrective surgery must be the result of the efforts of a number of specially trained and interested people.

Nurse. The nurse in the newborn nursery should be the one who recognizes the early abnormal signs produced by these congenital defects. The nurse who staffs the neonatal surgical postoperative unit is of

vital importance. Without good postoperative nursing, all the accuracy of diagnosis and surgical skill brought to bear in the infant's behalf goes unrewarded.

PEDIATRICIAN. It is he who must suspect the anomaly and recognize the urgent need for competent surgery. He must guide the infant through any associated medical problems and be ready as a consultant if unexpected complications of a medical nature occur during the postoperative period.

RADIOLOGIST, ANESTHESIOLOGIST, RESIDENT STAFF, SURGEON. All these specialists must be experienced and skilled in the problems presented by these neonatal emergencies. If any one of them is lacking or inadequate, the institution will fail in its care of the newborn infant who has a serious, but correctible, congenital defect.

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DIAGNOSIS OF CONGENITAL ANOMALIES INCOMPATIBLE WITH LIFE, BUT AMENABLE TO SURGICAL CORRECTION

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In neo-natal surgery we are dealing with infants suffering from conditions which will, if not corrected, cause death within a few days of birth. In hardly any other branch of surgery have there been such spectacular advances during the last 20 years as in the surgery of newborn infants. In the past the operative mortality rate was forbiddingly high, but during the last two decades there have been great advances, and today the prognosis is greatly improved.

In this short survey the diagnostic aspect of the various conditions is discussed, and treatment will be mentioned only in passing. It should, however, be pointed out that the great improvement in the operative mortality rate is due, not so much to better surgical technique, as to improvement in diagnosis, paediatric anaesthesia and especially in pre-operative and post-operative treatment.[17]

Early diagnosis is all-important if a high proportion of these infants is to be saved. It is, therefore, essential that every general practitioner, obstetrician, paediatrician and midwife be aware of the presenting symptoms of the various conditions and realise the extreme urgency of the problem. It is further important that all medical and nursing personnel who deal with newborn infants be trained in the first-aid treatment of surgical emergencies in neonates.

The various conditions causing surgical emergencies in the newborn period, mainly congenital malformations of the alimentary canal, are not common, and the average practitioner will see only a few in a lifetime. In this region of North West England, with a population of

about three million, practically all these infants are admitted to the Neo-natal Surgical Unit at Alder Hey Children's Hospital, Liverpool. We estimate that about 2 children per 1000 live births are admitted to the Unit.[13] It is, therefore, obvious that these conditions are so uncommon that, unless they are constantly remembered, the practitioner will easily delay the diagnosis. It is also obvious that if these children are admitted to various hospitals, the hospital staff will see only a few each year and will not gain enough experience. In order to improve the results, collection of the cases in special centres draining a large population is advisable. This will enable the surgeons, anaesthetists, pathologists, biochemists, radiologists and nurses working together as a team to become efficient in the management of these small infants.

The disadvantages of collecting newborn infants suffering from urgent surgical conditions in a few highly specialised centres is the delay which might occur by transporting these infants to the centre. However, in large countries such as the United States air travel expedites delivery of these infants to paediatric centres. It has been found that, provided these infants are accompanied by a nurse trained to supervise them, they will stand travel very well. All the infants in this area are transported by ambulance, in a carrycot or, if they are premature, in a portable incubator.

POSTERIOR CHOANAL ATRESIA

Congenital blocking of one of the posterior nares does not give rise to urgent symptoms at birth, but if both posterior nares are blocked by a membranous or cartilaginous or bony septum, the condition may produce severe symptoms endangering life in the neo-natal period (see also p. 1024).

Signs and Symptoms

Some newborn infants appear to be unable to realise that they can breathe through their mouth if both nares are blocked. Infants suffering from bilateral posterior choanal atresia will attempt to breathe through the nose, making more and more vigorous attempts at inspiration, sucking in their lips, but not opening their mouths. They quickly become cyanosed and will die unless the condition is recognised and their mouth forcibly opened. Air then rushes into the lungs, and the infants recover. Occasionally these infants breathe through their mouths for a time and then appear to forget, and another attack of suffocation develops. In those who survive, bilateral muco-purulent nasal discharge will be observed, and the children are unable to suck, since they cannot breathe during feeding. Unless, therefore, this condi-

tion is promptly diagnosed and the nasal block removed, these infants are very liable to die.

Diagnosis

The mouth of an infant who has suffered from an attack of suffocation, as described above, must be kept open with a prop. The condition can be diagnosed by passing a thin stiff rubber catheter along each nostril. In posterior choanal atresia the catheter will hit an obstruction

Fig. 1. Posterior choanal atresia. Iodised oil has been instilled into each nostril, with the child flat on his back. The oil is seen outlining the nasal cavities and does not run into the pharynx.

and cannot be passed into the pharynx. The condition can also be demonstrated by instilling some radio-opaque oil into each nostril in turn, whilst the child lies on his back, and then taking a radiographic picture. This will outline the nasal cavity and the posterior block (Fig. 1).

MICROGNATHIA

In this condition the mandible is grossly underdeveloped, giving the child a bird-like profile which is quite typical. Micrognathia is associ-

ated with cleft palate, but not with hare-lip. The tongue, being attached to the minute receding mandible, tends to fall back into the pharynx and may thus cause suffocation. In addition to producing acute dangerous respiratory embarrassment, the underdeveloped mandible makes feeding from the breast or bottle practically impossible (see also p. 1025).

Signs and Symptoms

The child's appearance makes the diagnosis obvious (Fig. 2). It is, therefore, surprising that the condition is occasionally not recognised. If the child is nursed lying on his back, the tongue will fall back into the pharynx, and sudden severe cyanosis coupled with frantic attempts

Fig. 2. Micrognathia.

to breathe shows that the respiratory passages are blocked. Turning the child on its face will relieve the attack. If an attempt is made to feed the child, he will splutter, cough and become cyanosed and distressed. These symptoms, rather similar to those of oesophageal atresia (see below), have occasionally led doctors to mistake micrognathia for oesophageal atresia.

OESOPHAGEAL ATRESIA

In this condition the oesophagus ends as a blind dilated pouch, usually in the upper part of the thorax. In over 90 per cent of the cases the distal oesophagus is present and connects the stomach with the trachea or a main bronchus, forming a tracheo-oesophageal fistula.

Only occasionally does the distal oesophagus end blindly without a communication with the respiratory tract (see also p. 1029).

Signs and Symptoms

The pathological anatomy described above explains the clinical findings. All mucus and saliva swallowed by the child will collect in the blind, proximal oesophageal pouch, and very soon mucus will flow back and dribble continuously out of the child's mouth; this is the most important presenting symptom. Occasionally the mucus may also overflow from the oesophageal pouch into the larynx, causing acute respiratory distress and cyanosis. This mainly occurs when the child's position is changed. Unfortunately all newborn infants tend to dribble mucus. The symptom may thus be overlooked, and a feed may be offered. If this happens, the first gulp of milk will fill the oesophageal pouch and overflow into the larynx and down the trachea. A weak baby may be drowned outright. A strong infant will react by coughing, spluttering, respiratory distress and cyanosis, and a fulminating pneumonia may develop.

The presence of a fistulous connection between the respiratory tract and the oesophagus can occasionally give rise to most alarming symptoms. Air is blown into the stomach under considerable pressure, especially when the infant cries. Ill-advised administration of oxygen through a face mask may also pump the oxygen straight into the stomach. The result will be extreme gaseous distension of the gastro-intestinal tract, causing pressure on the diaphragm and thus interfering with respiration.

Diagnosis

Oesophageal atresia is frequently associated with hydramnios in the mother, and infants whose mother suffered from hydramnios and who show the symptoms described above should be suspected of having oesophageal atresia until this have been disproved.[21] In suspected cases a rubber catheter should be passed down the child's pharynx. If the tube comes to a stop about 4 inches from the alveolar margin, the child is likely to suffer from oesophageal atresia. A soft rubber catheter may, however, curl up in the dilated blind oesophageal pouch (Fig. 3, A). A definitive diagnosis can be made only by radiography. One antero-posterior picture should be taken, and the blind oesophageal pouch may be outlined by instilling no more than 1 ml. of Lipiodol (Fig. 3, *B*), but is more safely made by passing a firm, radio-opaque rubber tube (Fig. 3, C). Air present in the stomach and intestine will prove the presence of a tracheo-oesophageal fistula.[6]

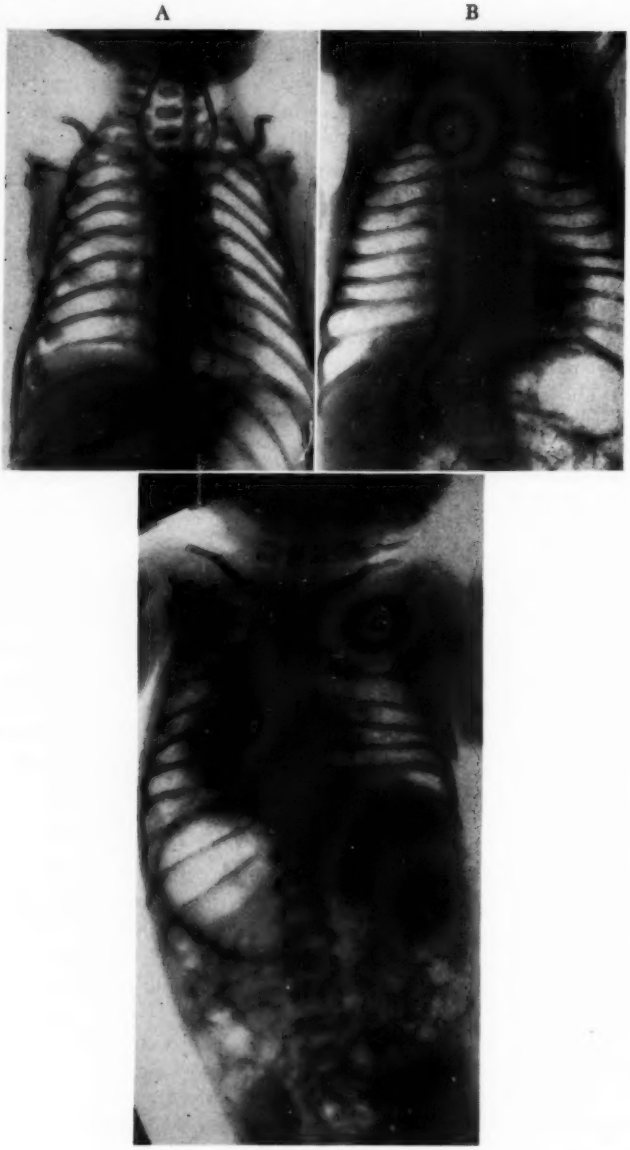

Fig. 3. Oesophageal atresia. *A*, A soft rubber catheter has been passed into the pharynx and is seen curled up in the blind oesophageal pouch. *B*, The tube has been passed into the blind oesophageal pouch, and 1 ml. of iodised oil has been instilled. *C*, A firm radio-opaque catheter has been passed into the blind pouch.

Oesophageal atresia is frequently associated with other serious malformations, especially intestinal atresia, rectal atresia and congenital malformations of the heart. Once the child has been diagnosed suffering from oesophageal atresia, the possibility of associated malformations should be suspected, and they should be looked for.

CONGENITAL INTESTINAL OBSTRUCTION

This is the most common cause for urgent operation in the neonatal period. From the prognostic point of view the various conditions giving rise to intestinal obstruction may be divided into two main groups. Those in which treatment necessitates opening of the intestine at operation carry a much higher mortality rate than those in which the intestine does not need to be opened.[19]

Signs and Symptoms

Vomiting is the most important symptom. It starts early and is profuse and sometimes projectile if the obstruction is high up in the intestinal tract; relatively late and less severe in low intestinal obstruction. Except in rare cases when the obstruction is above the ampulla of Vater, the vomitus is bile-stained.

With prolonged vomiting the initially bile-stained vomitus will become faeculent. In newborn infants faeculent vomiting occurs much more rapidly than in adults and older children and is not necessarily a sign of impending death. The gastric mucosa of newborn infants does not tolerate bile regurgitated from the intestines well, and severe gastritis will develop. In addition, the constant welling up of irritating stomach and intestinal juices will often produce a severe oesophagitis. It is, therefore, not surprising that a number of infants who have been vomiting for some time will bring up bright red blood.

Vomiting in newborn infants is dangerous. Not only will the infant quickly become dehydrated and lose large quantities of electrolytes, but also there is great danger that he will aspirate vomitus and either drown outright or die of pneumonia.

Abdominal Distension. Abdominal distension is minimal in high intestinal obstruction and occurs only in the epigastric region. In low intestinal obstruction it is generalised and often extreme. The grossly distended abdomen pushes the diaphragm upwards, and the infant's respiration, which is practically entirely diaphragmatic, will be seriously interfered with. Visible intestinal peristalsis or, in high obstruction, gastric peristalsis is occasionally observed.

Constipation. In theory no stools should be passed if the obstruction is complete. In fact, however, the passing of one or two small

meconium stools is not at all uncommon. The passing of a plug of greyish-white mucus is suggestive of an intestinal block. If the obstruction is only partial, stools will of course be passed, but they will be infrequent and small. The changing of meconium stools to milk stools will also be delayed.

Diagnosis

Any newborn infant who vomits bile-stained material shortly after birth should be assumed to have an intestinal obstruction until the diagnosis is disproved by radiography. A single anterior-posterior view with the baby held head up, feet down, is all that is necessary. In order to facilitate radiography and at the same time protect personnel from radiation, we bandage the infant in cruciform fashion on a mobile screen suspended in front of the radiographic plate, which slides between rails fixed to the screen (Fig. 4). The presence or absence of gas-filled intestine, the site and number of fluid levels will determine the diagnosis and give a rough estimation of the level of the obstruction (Fig. 5).

Fig. 4. X-ray screen, showing baby bandaged in a cruciform position in front of x-ray plate. Note that the head has to be bandaged to the screen in order to prevent it from falling forwards.

Fig. 5. Intestinal obstruction. A, Duodenal atresia. Note large fluid levels in the duodenum and in the stomach. There is no air in the abdomen. B, High jejunal atresia. There are only a few fluid levels in the stomach and upper coils of intestine. C, Low ileal atresia. There are multiple fluid levels all over the abdomen.

Certain types of intestinal obstruction in newborn infants need special consideration.

DUODENAL ATRESIA AND STENOSIS

In duodenal atresia the block is complete; in stenosis there is usually a membrane with a central hole or an extreme narrowing of the duodenum. This is the commonest site of intrinsic intestinal obstruction and differs from intrinsic intestinal obstruction of the more distal intestine by its being frequently associated with mongolism[3] and with congenital malformation of the heart.

In duodenal obstruction the epigastric region is usually full, and visible gastric peristalsis is commonly observed. In advanced cases vomiting is profuse and forceful and may be projectile. The newborn infant's stomach does not secrete much gastric juice during the first days of life. This may explain the curious fact that in duodenal atresia there is often not much vomiting for the first 24 to 48 hours.

About a quarter of the cases with duodenal atresia or stenosis are associated with an *annular pancreas*. In this malformation the head of the pancreas tightly surrounds the second part of the duodenum. In addition to the signs and symptoms described above, these patients may occasionally suffer from obstructive jaundice due to compression of the common bile duct.[14]

INTESTINAL ATRESIA

Atresia is uncommon in the small intestine and rare in the colon. Unlike oesophageal and duodenal atresia, atresia of the jejunum, ileum and colon is rarely associated with other severe malformations, and in our experience the prognosis is, therefore, quite favourable.

Diagnosis

The signs and symptoms of intestinal atresia are those of intestinal obstruction described above. In atresia of the lower small intestine abdominal distension may be extreme, the abdominal skin being stretched and shiny and respiration severely embarrassed.

Although meconium may be passed, as mentioned above, usually it does not contain squamous epithelial cells and lanugo hairs, swallowed by the foetus in utero. This is the basis of *Farber's test*.[5] The meconium is stained with gentian violet and examined under the microscope. Absence of epithelial cells and lanugo hair is supposed to indicate atresia. Unfortunately this test is not uncommonly fallacious, since it is based on the mistaken assumption that intestinal atresia occurs early in foetal life during the so-called solid state of the intestine. It has now been shown that most intestinal atresiae occur much later during foetal life and are secondary to intra-abdominal vascular catastrophes.[10, 12] It is, therefore, not uncommon to find lanugo hair and epithelial cells in the meconium in the presence of intestinal atresia.

DUPLICATIONS OF THE INTESTINE

Duplications of the intestine may give rise to obstruction in the neonatal period. In fact, obstruction is a more common symptom than massive intestinal bleeding (see also p. 1157) or perforation from a

peptic ulcer in the duplication, which is usually described as characteristic of the condition.

HIRSCHSPRUNG'S DISEASE

In our experience Hirschsprung's disease (see also p. 1150), formerly seen mainly in older infants and toddlers, is now practically always diagnosed during the neo-natal period. Many newborn infants with Hirschsprung's disease are admitted suffering from alarming symptoms of intestinal obstruction.

Signs and Symptoms

Abdominal distension and vomiting and the absence of meconium stools are the presenting symptoms. After rectal examination or the giving of a small enema it is not uncommon if a plug of greyish mucus is passed per rectum followed by masses of soft meconium. The child will then often deflate itself and vomiting cease. This is very suggestive of the so-called meconium plug syndrome.[4] Frequently the child is now discharged, only to return after a few days with recurrent subacute intestinal obstruction. Occasionally the symptoms of intestinal obstruction are more acute. Profuse vomiting will quickly lead to severe dehydration, and enemata are returned without the infant's passing any flatus. These are the cases with a long aganglionic segment of colon, often affecting the whole of the sigmoid colon.[8]

Diagnosis

A radio-opaque enema, preferably an iodine-containing watery medium, will outline the colon, and, in about one third of newborn infants suffering from Hirschsprung's disease, the typical contracted rectum and variable length of lower colon with dilatation of the proximal colon will be observed.[21] Unfortunately it usually takes some little time for the proximal gut to dilate, and frequently the radiologic picture shows a gradual funnel-shaped widening of the proximal bowel. It is, however, of the greatest importance to make an accurate diagnosis, since treatment must not be delayed if unnecessary fatalities are to be avoided. A rectal biopsy should, therefore, be carried out in all suspected cases. Microscopic examination of the excised rectal muscle and mucosa will determine the presence or absence of ganglion cells.

MECONIUM ILEUS

Meconium ileus is one aspect of a generalised congenital malformation of the mucus-secreting glands of the body, mucoviscidosis.[2] It is due

to fibro-cystic disease of the pancreas causing failure of secretion of pancreatic enzymes. Without pancreatic enzymes the meconium becomes the consistency of chewing gum, sticking to the walls of the intestine like glue. A mechanical obstruction due to blocking of the intestinal lumen by sticky meconium thus results. The obstruction usually occurs in the lower ileum or colon where the meconium forms small, sticky, rabbity lumps. The intestine proximal to this block becomes dilated and filled with fluid and air.

Signs and Symptoms

Apart from the signs and symptoms of low intestinal obstruction, meconium ileus presents some special features worthy of mention. Not infrequently the infant looks grey, or there is slight, dusky cyanosis. There may already be evidence of beginning broncho-pneumonia. Broncho-pneumonia attacks secondary to fibro-cystic disease of the lung frequently occur and often follow a fulminating course. They are the main causes of death.

Some infants pass no meconium at all, others pass a few dry, greyish pellets, and again in others a quantity of black, sticky meconium may be evacuated after a rectal examination has been performed. The abdominal distension is that of low intestinal obstruction. Occasion-

Fig. 6. Meconium ileus. Note the enormously dilated intestine and "marbled" shadows in the right iliac fossa.

ally it is possible to palpate strings of bead-like, firm rabbity stools in the colon, especially in the right iliac fossa.

Diagnosis

A straight radiographic picture in the upright position shows dilated coils of intestine. In addition, it is not uncommon to observe shadows which on close inspection have a marbled appearance due to the presence of numerous minute gas bubbles (Fig. 6). These "marble" shadows are due to sticky meconium interspersed with air bubbles, which have been squeezed into it by vigorous peristaltic waves. They are supposed to be diagnostic of meconium ileus,[11] but are occasionally also seen in other types of intestinal obstruction of the newborn.

When testing the meconium for the presence of trypsin, none will be discovered, and there will also be an absence of pancreatic enzymes in the duodenal juice. This is, however, not pathognomonic of meconium ileus, since it is not infrequently observed in newborn infants, especially in infants suffering from other types of intestinal obstruction.

Cystic fibrosis of the pancreas is a familial condition, and a history of other members of the family dying of intestinal obstruction early in infancy or suffering from bronchiectasis is, therefore, suggestive.[1]

PERITONITIS

Peritonitis (see also p. 1079) is not uncommon in the neo-natal period and may be of two types.

Meconium Peritonitis

If an intestinal perforation occurs in utero, sterile meconium will escape into the peritoneal cavity and cause an aseptic chemical peritonitis. Perforation occurs proximal to an intestinal obstruction, and meconium peritonitis is, therefore, commonly found in association with such conditions as intestinal atresia, meconium ileus or volvulus. Meconium peritonitis may, however, also occur when there is no intestinal block. If the perforation is still patent at birth, an infective peritonitis will soon result. In many cases, however, the perforation closes in utero and there is no sign of it at birth.[7] Dense adhesions form in the peritoneum as a result of this peritonitis and cause kinking, strangulation, volvulus and secondary atresia of the intestine.

Bacterial Peritonitis

This may be due to an intra-uterine perforation remaining patent after birth, a perforation occurring after birth (i.e., perforating peptic ulcer,

or trauma), or to the presence of gangrenous gut in the abdomen (usually secondary to strangulation), or may be haematogenous.

Signs and Symptoms

Infants suffering from peritonitis are usually, but not invariably, very ill and are admitted in a shocked condition. The abdomen is often enormously distended with dilated veins visible beneath the abdominal skin. Oedema of the abdominal wall may be conspicuous, especially in

Fig. 7. Infant with large subphrenic abscess originating from an infected umbilicus. The epigastric region above the umbilicus is bulging because of the underlying abscess.

infective peritonitis. The child looks grey, respiration is rapid, shallow and occasionally grunting, and there may be a tinge of cyanosis. In meconium peritonitis the abdomen is often filled with large amounts of fluid and will, therefore, be dull to percussion, and a fluid thrill may be elicited. If the perforation is still patent at birth, there will be an intra-peritoneal air bubble, which shifts with posture and can sometimes be demonstrated by percussion. There is paralytic ileus, and bowel sounds will be absent on auscultation.[16]

In cases with infective peritonitis there may be evidence of septi-

caemia (jaundice, petechiae). The umbilicus should always be carefully inspected, since it is the main portal of entry for infection. A red, weeping or frankly purulent umbilicus should be viewed with suspicion. Frequently there is an attempt at localisation of the infection in the peritoneal cavity, and large abscesses may form which can be palpated or may even be visible on inspection (Fig. 7). Stools are passed, loose, mixed with mucus and pus and evil-smelling.

Diagnosis

Radiography is usually diagnostic. In meconium peritonitis multiple irregular calcified shadows may be seen as the intra-peritoneal meconium masses rapidly calcify. This calcification must be differentiated from calcification of the meconium in the lumen of the intestine, which may occur in meconium ileus, or rarely for no apparent reason.[18]

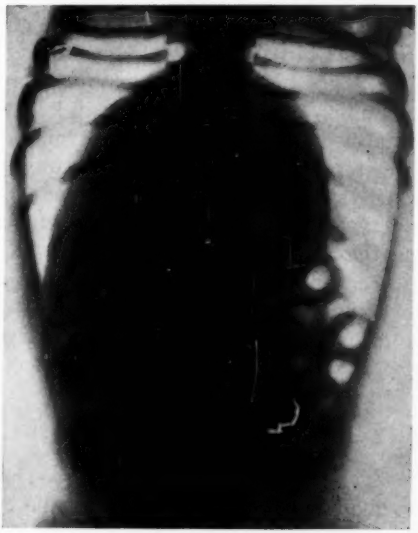

Fig. 8. Pneumo-peritoneum secondary to perforation of the intestine. A large amount of air can be seen beneath the diaphragm.

A pneumo-peritoneum is clearly visible on radiography. It may be very large, distending the abdominal cavity and pushing the diaphragm upwards (Fig. 8).

VOLVULUS OF THE MIDGUT

The normal foetal rotations of the midgut are so complicated that it is not surprising that many abnormalities occur. In the most common

type the caecum fails to descend into the right iliac fossa and remains just beneath the liver. In this position it develops new attachments, and fibrous bands pass from the colon to the liver and right lateral abdominal wall across the second part of the duodenum. The duodeno-jejunal and ileo-caecal junctions come to lie side by side. There is then no attachment of the mesentery to the posterior abdominal wall, and the whole of the small intestine is suspended from a narrow pedicle and frequently rotates, causing a volvulus of the midgut and hence a closed loop obstruction, which may strangulate and become gangrenous.[9] (See also p. 1158.)

Signs and Symptoms

The condition frequently, although not invariably, gives rise to acute symptoms shortly after birth. Symptoms are produced in two ways. Firstly, incomplete duodenal obstruction may be caused by the bands crossing the duodenum, and frequent, profuse, bile-stained, occasionally projectile vomiting will be the presenting symptom. Secondly, the twisting and untwisting itself may cause symptoms. There may, therefore, be occasionally a history of periodic attacks of vomiting, abdominal distension and constipation, with resolutions without treatment. If strangulation and gangrene occur, there will be, in addition, abdominal tenderness and shock and signs of peritonitis. It is surprising that in newborn infants shock develops relatively late, and we have frequently seen children admitted in a good general condition who on laparotomy, to our surprise, had gangrenous intestine.

Diagnosis

Radiography of the abdomen usually shows a large stomach air bubble and frequently a small bubble in the first part of the duodenum. There is little gas in the rest of the abdomen. What gas bubbles there are, are usually small and bunched up in the centre of the abdominal field.

STRANGULATED INGUINAL HERNIA

Signs and Symptoms

This condition is mentioned only because it occurs frequently during the neo-natal period. The symptoms are typical of intestinal obstruction, and the presence of a small lump in the groin of a fat infant may be overlooked. Strangulation of an inguinal hernia is uncommon in infants, and the incarcerated hernia is, therefore, usually not tender. It goes without saying that in all infants with signs of intestinal obstruction, the groins should be carefully examined. (See also p. 1129.)

RECTAL ATRESIA

There are two main types of rectal atresia. In high rectal atresia the rectum ends blindly, usually at the level of the peritoneal reflection. The blind pouch nearly always communicates with the base of the bladder or posterior urethra. The levator ani is often poorly developed or absent. In the variety of low atresia the rectal pouch ends blindly just underneath the skin. This variety is also associated with fistulae, which may open into the perineum anteriorly to the anal dimple, the vagina or anywhere along the perineal raphe. The levator ani is present.

Signs and Symptoms

Inspection of the perineum after birth should immediately reveal that there is no normal anal opening. It is, therefore, surprising that the condition is frequently overlooked and discovered only when the symptoms of low intestinal obstruction have supervened. Mistakes may be made because of the presence of a fistula; small amounts of meconium may be passed through this, and the diapers may show meconium staining.

On inspection of the perineum there is usually an anal dimple or a patch or strand of skin at the site of the anal orifice (Fig. 9). In low

Fig. 9. Low rectal atresia. The anal orifice is covered by a hypertrophied speck of meconium which extrudes from a minute perineal fistula, shown by a probe.

rectal atresia the perineal skin will bulge when the child is crying, and rarely there is a thin, translucent membrane at the site of the anus, through which greenish-black meconium is seen to bulge. The minute perineal or vaginal fistulous opening is often discovered by noticing a small speck of meconium. Occasionally a thin black fistulous track filled with meconium and covered by translucent skin is seen coursing forward in the midline along the perineal raphe opening at the perineal-scrotal junction or, more rarely, anywhere along the scrotal raphe or even the ventral surface of the penis.

Diagnosis

The presence of a low rectal atresia can usually be diagnosed by inspection and palpation as described above. The level of high rectal atresia may be estimated by a lateral radiograph of the infant's abdomen and pelvis after the infant has been suspended for three minutes upside down to allow gas to rise into the blind rectal pouch[23] (Fig. 10). Unfortunately this estimation is quite unreliable, since the thick meconium

Fig. 10. Lateral radiograph of an infant with high rectal atresia. The infant was hung upside down for 3 minutes, allowing gas to rise into the blind rectal pouch. The level of the perineum is indicated by a metal marker.

in the blind pouch is often only partially replaced by gas, and the level of the pouch appears, therefore, much higher than it in fact is. Absence of sacral vertebrae or deformities of the sacrum have a bad prognostic significance, since they are associated with maldevelopment or absent levator ani; hence continence after a pull-through operation will be poor.

EXOMPHALOS (OMPHALOCOELE)

In this condition there is a larger or small gap in the abdominal wall at the base of the umbilical cord. The intra-abdominal organs

Fig. 11. Omphalocoele. A, Small omphalocoele, or herniation into the cord. *B*, Large omphalocoele. *C*, Intrauterine rupture of omphalocoele. The shortened coils of intestine grossly thickened by oedema are seen protruding from the abdominal cavity.

protrude through this gap and are covered only by the thin, translucent amniotic membrane (see also p. 1101).

Signs and Symptoms

If the herniation is small, only a knuckle of intestine may protrude into the base of the umbilical cord and may be overlooked (Fig. 11, A). In the large types of omphalocoele most of the intestine and often part of the liver will protrude through a wide circular gap, and the abdominal cavity will be empty and contracted (Fig. 11, *B*). Diagnosis is obvious on inspection.

If treatment is delayed and the amniotic membrane is allowed to dry and become infected, cracks will occur in it, and bacteria will penetrate into the peritoneum to cause peritonitis. Alternatively, the sac may burst and evisceration occur. Occasionally the exomphalos ruptures during foetal life, and the child is born with eviscerated intestine (Fig. 11, C). In this case the gut is usually very short, but the walls are enormously thickened by oedema. Ruptured exomphalos is almost always fatal.

Diagnosis

Although the diagnosis is obvious, a radiographic picture of the abdomen and chest should always be taken in order to discover possible associated malformations of the heart or intestinal tract, which are not at all uncommon.

DIAPHRAGMATIC HERNIA

The only varieties of diaphragmatic hernia (see also pp. 1038, 1155) giving rise to urgent symptoms in the neo-natal period are those through a patent pleuro-peritoneal canal, a hernia of Bochdalek, or through an area of congenital absence of one of the anlagen of the diaphragm. The former occurs much more commonly on the left than on the right side. If there is a gap in the diaphragm, the intestines will herniate into the chest. The left lung will, therefore, fail to expand at birth, and the mediastinum will be pushed over to the opposite side and compress the right lung. Frequently practically the whole of the small intestine, stomach, spleen, part of the large intestine and the liver are in the chest.

Signs and Symptoms

Severe respiratory distress occurs directly after birth. Difficulty in breathing and cyanosis increase as the child swallows air and the stomach

and intestine distend. Dyspnoea is often so acute that unless an endotracheal tube is passed immediately and the child is kept on positive pressure inhalation of oxygen, it will quickly succumb. Positive pressure oxygen by mask further inflates the stomach and may produce death promptly.

Diagnosis

Hyperresonance on percussing the affected side of the chest, displaced apex beat and the presence of bowel sounds on auscultating the chest,

Fig. 12. Diaphragmatic hernia through a left-sided patent pleuro-peritoneal canal. The left pleural cavity is filled with intestine; the heart and mediastinum are grossly displaced to the right. The only part of the gastro-intestinal tract is the stomach, which can be seen outlined with air.

coupled with a scaphoid, empty abdomen, will usually make the diagnosis obvious. On radiography numerous typical bowel shadows will be seen in the affected side of the chest, and shifting of the mediastinum will be noticed (Fig. 12).

In newborn infants diaphragmatic herniae rarely strangulate. Shock, uniform dullness on percussing the affected side of the chest and the passage of blood per rectum may suggest this complication.[15]

CONCLUSION

In this short paper it is possible only to describe briefly the more important signs and symptoms and to confine the discussion to the more important of the many congenital abnormalities which endanger the newborn infant's life.

If doctors and nurses are aware of the main diagnostic points and are constantly reminded of the various malformations occurring in newborn infants, admission to hospital should not be unduly delayed. By and large this aim has now been realised in this region, with the result that over 80 per cent of full-term infants suffering from such conditions as oesophageal and intestinal atresia, volvulus, exomphalos, and others, survive operation, although there are a few conditions such as meconium ileus and peritonitis in which the operative mortality rate is still high.

Infants with multiple malformations each one of which endangers the child's life will only rarely survive operative correction, but most of these infants are so grossly deformed that their death does not appear to be of much consequence. The majority of infants, however, who survive operation for a single severe malformation develop into perfectly normal children.

There remains the problem of the premature infant suffering from any of the conditions described above. In the grossly premature infant diagnosis is often delayed, because his reactions are less vigorous and the signs and symptoms are vague. On the other hand, pre-operative and post-operative management is very difficult, and the mortality rate is high.[20] Future developments in paediatric surgery will undoubtedly aim to overcome these special problems.

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PREPARATION OF THE CHILD FOR SURGERY

Responsibility of the Referring Physician

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The successful outcome of an operation performed on a child depends not only upon the skill of the surgeon, but also upon the work of a team of individuals concerned with the total care of the child. Included in this team are the referring physician, admitting clerks, internes and residents, nurses, ward aides, "toy ladies," anaesthetists, the surgeon, and the parents of the child.

An important member of this team is the referring physician, because, of all the members, he alone knows the child, the parents and the family situation, and from previous experience he is likely to know how the parents will behave under stress. Because the child and the parents know and trust their doctor, they are more likely to reveal their worries and fears to him than to strangers. Both from a physical point of view and from an emotional standpoint, the referring doctor is in a better position to help prepare the child and the parents for surgery than are any of the others who will be concerned with the care of the child once he is in hospital.

Depending upon the age of the child, and upon whether the operation to be performed is elective or emergency, the role of the referring physician in the pre-operative preparation of the patient will vary to a great degree.

URGENT SURGERY IN THE NEWBORN

When a baby is born with a serious abnormality requiring urgent surgical treatment, the young parents are usually completely overcome by the tragedy that has fallen their way. They will be absolutely incapable of making any rational decisions about the care of their child, and all

Fig. 13. Incubator* used for transporting infants at the Winnipeg Children's Hospital. The heating unit can be plugged into the cigarette lighter socket of an automobile. Oxygen (if indicated) is supplied by a portable tank.

decisions and arrangements must be made by the referring physician.

Having contacted the surgeon and informed him of the baby's condition, the referring physician must then make arrangements to transfer the infant from the newborn nursery to a surgical centre equipped to handle major surgery in this age group. If the maternity hospital and the surgical centre are close by, transfer can usually be made easily in a warm car, the blanket-wrapped baby being carried by a nurse. If, as often happens, the infant has to be transported some distance, then, as Bishop[1] has described, more satisfactory arrangements must be made.

Many hospitals have portable incubators which can be warmed as required, and these are ideal (Fig. 13). Every effort should be made to maintain the small infant's body temperature at a normal or slightly sub-normal level. Even under the most severe climatic conditions the tendency is to overwarm the child and to produce an elevation in body temperature which, by increasing the metabolic demands, may be detrimental to the seriously ill infant.

During transportation, oxygen should be available for use if indicated, but it should not be routinely used.

If the abnormality involves the gastro-intestinal tract, so that vomiting, with danger of aspiration, or so that increasing abdominal distension may be a serious complication, then an indwelling nasal catheter

* The Accli-Bator Portable Incubator, Model 510, manufactured by the Accli-Bator Company, 830 North La Brea Ave., Los Angeles 38, Calif.

should be inserted, and oesophageal or gastric contents aspirated. During transportation the nurse to accompany the infant should be instructed to aspirate the catheter intermittently, using a syringe.

To guard against complicating infection, particularly pulmonary infection, an antibiotic should be administered to the child before transfer. Because transient hypo-prothrombinaemia of the newborn may complicate the operation, it is wise for the referring physician to administer to the baby a small dose of vitamin K (not exceeding 2.5 mg.).

A competent nurse should accompany the child, and, if possible, the father should go too, so that he can fill out the necessary admission forms and sign the operative permission. If the father cannot accompany the baby, then it is essential that the referring doctor send this necessary information and an operative permission form, signed by either the mother or the father.

Any roentgenograms taken on the infant should be sent to the paediatric surgical centre, since time-consuming repetition of these diagnostic studies may be avoided. In all instances the referring doctor must send along a brief history containing essential information about the family history of other congenital abnormalities, the pregnancy of the mother, the delivery of the child, the post-natal course with particular reference to episodes of respiratory distress, the amount and frequency of vomiting, the type of vomitus, the number of voidings, and the number and type of meconium stools. Any laboratory data must be accurately recorded, and, again, the supplying of this information may save valuable time once the baby arrives at the surgical centre. An account of treatment, intravenous fluids, drugs, and so on, should also be included, since further care of the child may be greatly influenced by this information.

Finally, in the note to the surgeon, the referring physician should suggest the best arrangement by which the surgeon can inform the physician and the parents of the outcome of the operation and the progress of the child.

The referring physician must make every effort to bolster the morale of the mother, so that she may make a prompt recovery, and be able to care for her baby when he is subsequently discharged from the paediatric centre.

ELECTIVE SURGERY IN INFANTS AND SMALL CHILDREN

When an infant or a small child under the age of four requires an elective surgical procedure, little or nothing can be done to prepare the child for hospitalization and surgery, from an emotional aspect. The small child cannot understand, but the child over two years of age should be informed that he must stay in hospital. He should be told

that the doctors and all the people in the hospital will make him well again as quickly as they can, and that he will then go home. The referring physician should explain the situation to the parents, reassure them as to the qualifications of the surgeon, and attempt to resolve their fears and doubts so that these will not be communicated to the poorly understanding child.

From a physical aspect, however, the referring physician has a definite responsibility. A thorough physical examination should be performed, so that any intercurrent infection or previously undiscovered abnormality may be detected. Before operation is performed, any infection should be treated and cured. A diaper rash should be cleared up, and any anaemia corrected. Pre-existing dietary insufficiency, if present, should be determined and, if possible, relieved before the time of elective surgery. The immunization of the child should be reviewed and, if necessary, be brought up to date well ahead of the time of hospitalization.

Information about drug sensitivity or food allergy and any familial disease should be communicated to the surgeon, so as to avoid complications during hospitalisation. Because of the ever-present danger of cross-infection in the hospital, every effort should be made to make the period of hospitalisation as short as possible.

If the infant is being breast-fed, the referring physician, in consultation with the surgeon, should make arrangements for the mother to continue breast-feeding the child while in hospital, or, if this is impossible, for her to pump her breasts at home and have the milk brought to the hospital each day.

It is also the responsibility of the referring physician to check on the health of other members of the family, and to treat any infection with which the patient may come in contact.

EMERGENCY SURGERY IN OLDER CHILDREN

Obviously, in an emergency situation, there is little or no time for other than the essential preparation of the ill child for the operation. Most of this will be the responsibility of the surgeon himself.

The referring physician, however, should introduce the surgeon to the child and to the parents. The child should be told that he is going to have an operation, and that the surgeon will fix the pain or trouble and will make him well again.

To the parents, anxious and sometimes almost terrified, the referring physician should briefly outline the situation, and reassure them that the surgeon has handled this sort of illness many times before, and can be relied upon to treat the child to the best of his ability.

The referring physician should tell the surgeon essential information about the family, their conduct under previous stress, their worries and

fears, so that when the surgeon talks to the parents, he may have this background of knowledge in reassuring them about the operation to be performed on their child. Their financial situation should be briefly outlined to the surgeon. Many parents in an emergency situation request private accommodation and special duty nurses, and state that expense is of no consideration. The referring physician, knowing the family's financial situation, can see to it that good care is provided, but not unnecessary care which will indebt the family far beyond their financial resources.

Any allergies or sensitivities of the child should be noted on the chart. The immunisation to date may be of importance and should be carefully recorded. History of exposure to contagious disease should be determined, and, if the child has been exposed, arrangements should be made for him to be treated in isolation.

When the child has been taken to the operating room, the parents should be told where they are to wait and how they will be informed about the operation and the post-operative progress of their child. If the hospital has a post-operative recovery room, the parents should be told in advance that their child will not immediately return to his bed. If the family circumstances do not permit the parents to wait during the operation, the referring physician and surgeon should arrange how the parents will be informed of the results of the operation, and who will do it.

Acute laryngo-tracheo-bronchitis constitutes a possible surgical emergency deserving special consideration. This disease occurs most frequently in the winter months, and the respiratory difficulty often becomes greatest in the small hours of the night. If the physician, at the child's house, suspects that the child may require a tracheotomy, he should immediately notify the hospital to have all emergency equipment in readiness. He also should arrange for the surgeon to be at the hospital to meet the child on arrival. The readying of the child for the trip, and the drive through the cold night air, frequently aggravate the respiratory obstruction, so that a child, previously only moderately distressed, may arrive at the hospital cyanosed and desperately in need of tracheotomy. With the operating team in readiness, and the surgeon prepared, this lifesaving procedure can be immediately performed.

ELECTIVE SURGERY IN OLDER CHILDREN

Children over four years of age who must have elective surgery can be helped both from a physical and particularly from an emotional or psychic aspect by careful pre-operative preparation.

As mentioned before, with young children, the physical condition of the older child should be carefully evaluated and any deficiencies corrected before operation.

Prugh[2, 3] has reported that over one-third of children studied, showed some reaction to hospitalisation for either surgical or medical illness, and this reaction persisted for at least three months after discharge from hospital. In actual fact, the proportion of children showing psychic disturbance as a result of hospitalisation or surgery is likely higher than this figure, but minor post-operative behaviour problems may not be mentioned by the parents to their doctor. Much can be done to prevent these emotional problems by pre-operative preparation, and much of this is the responsibility of the referring physician or paediatrician.

The child, after having been examined, and the need for elective surgery determined, should be removed from the doctor's office to be entertained by the nurse. The situation should then be clearly and concisely explained to the parents. This explanation must not be too detailed, since details will confuse or alarm the parents, but it should be sufficiently clear so that they can fully understand the need for surgery, and what is to be done. Doubtful, anxious and worried parents will communicate their fears to the child, and every effort must be taken to reassure the parents.

The referring physician should instruct the parents in the psychological preparation of their child for hospitalisation. Most children's hospitals have booklets for parents, giving them advice on this, and it is the responsibility of the referring doctor to see that the parents receive this information.

If the hospital does not have a booklet of this type, it is the responsibility of the physician to explain to the parents the hospital's admitting routine, the time for admission, the type of accommodation available, the articles of clothing to be taken to the hospital, the visiting hours, and so forth, and he should invite the parents to ask him any questions they might have.

If the operation is to be performed within a few days of the office visit, the older child can be told by the doctor that the operation is to be performed. If it is to be delayed for several weeks, the child should not be told of the proposed operation until a few days in advance of hospitalisation. This should be done by the parents. Under no circumstances should the child not be told that he is going to the hospital. Certainly he should not be lied to and told that he is going to a circus or for some other special treat. Fear of the unknown is prominent in anyone, and children are no exception. Usually a careful, calm explanation by the parents or the doctor of the necessity for hospitalisation and what it involves will benefit the child more than any amount of bribery or cajolery.

At the Children's Hospital in Winnipeg, every Saturday morning, a motion picture film is shown.* The parents and their child (over four

* L. E. Hackworth: A True Story about Hospitals. Children's Hospital, Los Angeles, Calif.

years of age) are invited to the hospital on the Saturday of the week before the scheduled operation. They are taken on a brief tour of the hospital, including the admitting office and the wards, and are shown the playrooms, the waiting rooms and the cafeteria. Any questions they have about the admitting routine, visiting hours, and so on, are answered by the staff nurse in charge of the tour. They are then shown the motion picture film, which clearly outlines what will be done for the child after his arrival at the hospital. Any further questions are answered, the parents are served coffee, and the children are given a dish of ice-cream.

Although not all parents take the opportunity of seeing this film, it is our definite impression that those families who do, benefit greatly. Their child frequently arrives at the hospital for admission almost in anticipation of the experience, rather than worried and fearful of having to face the unknown.

Colouring books or comics which describe the hospital and its routines are useful, but care must be taken that these outline the methods used in the hospital to which the child is to be admitted, because serious discrepancies between those described in the books and those of the hospital may confuse, rather than reassure, the child.

Playing games of nurse or doctor at home before hospitalisation may also help in the pre-operative preparation of the child, but the success of these games depends almost entirely on the time available to the parents and their intelligence in making the games worth while. A neighbour child, allowed to participate in the game, telling stories of needles, enemas and other unpleasant procedures, will in a few minutes undo any good that the games might otherwise have accomplished.

Under no circumstances should the referring doctor misinform the parents or the child with the hope of thus reassuring them. To mislead them that the child will not receive any needles, will not have any discomfort, that the mother can stay all night with her child, and that the anaesthetic will be given in the child's own room, when the hospital has not the facilities to carry out these promises, will lead to distrust and increased anxiety, rather than to reassurance.

Above all else, a kindly manner, a common-sense truthful approach, and a clear and unhurried explanation of the need for surgery will be of great benefit to the child and to the parents in avoiding post-operative emotional upsets.

DISCUSSION

Most of the foregoing has been written with the assumption that the child will be admitted either to a children's hospital or to a general hospital with a large paediatric service, and that the surgeon concerned will be at least familiar with, if not an expert in, the care of infants and children. If, however, these ideal circumstances are not available,

then the referring physician or paediatrician must assume a far greater responsibility.

Not only must he do what has already been outlined, but, in addition, he must assume the full pre-operative care of the infant or child. He must leave all the orders for medication, and may be required personally to manage the pre-operative fluid therapy if dehydration or electrolyte imbalance is present. Only after the child has been improved as much as is possible should the operation be performed.

When the surgeon has had little or no experience in the care of diminutive patients, it is well for the referring paediatrician and the surgeon to decide, in advance of the operation, the exact responsibilities each will assume in the care of the baby. By this previously arranged division of responsibility, conflicting orders written by each doctor, and the resulting confusion to which this can lead, may thus be avoided.

SUMMARY

The responsibility of the referring physician or paediatrician in the preparation of the infant or child for hospital has been reviewed. Since this varies greatly, depending upon the age of the child and upon whether the operation is urgent or elective, the subject matter has been considered under four headings: urgent surgery in the newborn; elective surgery in infants and small children; emergency surgery in older children; and elective surgery in older children.

An attempt has been made to describe the preparation of the child (and of his parents) for surgery, both from the physical and emotional aspects.

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TIMING IN PEDIATRIC SURGERY

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To formulate rules for timing in pediatric surgery is simpler in theory than in practice. Through necessity any rules will be subject to many variables which can only be evaluated individually. But for purposes of teaching, rules will provide a framework on which to elaborate. Timing not only involves the optimal age for a specific procedure, but also includes a number of other factors which merit careful attention.

THE ELECTIVE OPERATION

Safety

This must always be a prime objective, and any operative procedure deemed elective should be carried out when the infant or child is best able to withstand it. Undue pressure from a family should not be allowed to influence a surgeon, and the pediatrician can do much to help by allowing the surgeon to make an open decision. Whatever influence the pediatrician wishes to bring to bear should be directed to the surgeon himself and not through the family. By contrast, surgeons can give weight to the opinion of the referring pediatrician, who has had the opportunity to understand the behavior pattern of the individual child from longer contact.

Hemoglobin Levels

A hemoglobin level below normal may result in deficient oxygen transport under conditions of stress. This anemia presents an additional

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hazard to the child undergoing anesthesia. The risk may be small, but if the stress is unusual—perhaps severe laryngospasm during induction—the patient will incur a greater risk of injury such as brain damage. An arbitrary level of 10 gm. of hemoglobin per 100 ml. of blood is accepted as the minimum required. Below this level two courses are available—to correct the usual iron deficiency anemia by administering iron for a sufficient time or to raise the level by means of blood transfusion. For the simple anemia the first course is preferred; the latter is less desirable, since it incurs other risks, but may have to be used when conditions demand. When the anemia itself merits blood transfusion, the hemoglobin level should be raised to at least 10 gm., but 24 to 48 hours should elapse after the blood administration before any operative procedure.

Upper Respiratory Tract Infections

Children with colds try the patience of referring physician and surgeon alike, but an incipient cold is ample justification for delaying an elective operation which requires general anesthesia. To sit all night with a child who is struggling for breath because of a cold complicated by an anesthetic cannot easily be forgotten. A delay of one or two weeks is often adequate to permit the patient to tolerate the procedure in safety. Further delay invites the risk of a fresh cold. The snuffly nose with a history of a chronic mild respiratory infection which lasts all winter presents little danger and rarely warrants delay.

Emotional Problems

It might be stated, from the child's point of view, that there is no good time for admission to a hospital. Nevertheless the majority of infants and children tolerate this psychic trauma extraordinarily well. The concept of admitting the mother with the child may spread and perhaps will eliminate some problems, though it often raises others for the medical personnel.

Separation problems are more evident from 18 months to 4 years of age, and this age group will benefit most from careful preparation. Members of the surgical staff should take time to explain, within the child's scope, what he will have to expect in the form of insults to his person, injections and postoperative discomfort. Parents, too, can do a great deal to prepare their child for the hospital admission, but they should neither make the prospect too frightening nor paint too rosy a picture. The referring pediatrician who knows the child well can help the surgeon immeasurably by advising him of the patient who needs unusually careful handling. Lengthy procrastination is seldom

needed, and the surgeon should generally choose the time for the operation, since he will have to cope with any complications resulting from delay. (See also p. 979.)

THE URGENT SURGICAL PROCEDURE

Timing for these patients is more a question of minutes and hours than days. Nevertheless many aspects must be considered in order to bring the patient to the point at which surgical intervention will be best tolerated.

Blood Transfusion

The necessity for blood transfusion in a seriously ill child may be overlooked in the anxiety to deal rapidly with the surgical problem. Acute appendicitis with peritonitis, or intestinal obstruction may be cited as examples. Induction of anesthesia and surgical trauma both combine to alter the relative circulating volume and may produce a precipitate fall in blood pressure in the hypovolemic patient. Nor is the hemoglobin always a reliable examination, since hemoconcentration may give a false sense of security. If any doubt exists, then blood should be cross-matched and available for the procedure with a needle or plastic cannula in a vein, for rapid administration.

An inadequate hemoglobin concentration should be treated with transfusion before operation if possible, and certainly blood transfusion must be started at the time of the incision.

There is a temptation to attack any lesion causing hemorrhage as fast as possible. However, this must be tempered by the absolute necessity to control any shock first. Rarely, traumatic injury may justify immediate attack on the bleeding area when it is readily accessible, but, in general, blood transfusion should be started before anesthetizing any child who has sustained a significant blood loss.

Electrolytes

The advent of microchemical techniques has been valuable for infant surgery. A brief delay to obtain information about the serum levels of sodium, potassium, chloride and carbon dioxide is justified in any surgical patient with clinical evidence of depletion of electrolytes. Particularly for the patient with intestinal obstruction, this information can determine the preoperative preparation needed and provides a base line for postoperative electrolyte and fluid administration. With the exception of the patient with volvulus of the gut, a delay of 12 to 24 hours to correct severe electrolyte deficits is often essential.

Respiratory Tract Infections

Urgent surgery presupposes no lengthy delay, and respiratory infections must be handled by protecting the patient with the means available. This may involve the use of unusual anesthetic techniques for children and control of secondary respiratory infection with antibiotics. Postoperatively an atmosphere with high humidity should be maintained until the risk of airway complications has been averted.

Other Factors

Numerous other considerations have to be taken into account. The maturity of infants is often a deciding factor in the type of operative procedure planned. If a simpler but temporary maneuver can be done with significantly less risk, then it should be favored. The presence of a second anomaly requiring surgical attention demands skill in the timing and planning of procedures to ensure the best chance for survival. A single, more extensive operation will probably be better tolerated than two major operations within a few days, but as it is not always possible to attack the problems with a single operative exposure, then the more life-endangering anomaly should be dealt with first. The importance of careful preoperative diagnosis is vital, for, without it, planning is impossible.

In preparing any patient for an operation there comes a time when the preoperative therapy has gained all possible advantages, and this is the time for the surgeon to act. Beyond this point, efforts invoke the law of diminishing returns—correction effected is outweighed by the accruing problems. There is no rule whereby this time can be pinpointed, and only by frequent observation of the patient and reassessment of the clinical status can the surgeon determine the ideal time.

SPECIFIC PROBLEMS

The timing of any elective operative procedure depends upon the facilities available for diagnosis, the anesthetic and surgical skills which can be brought to bear upon the problem, and the facilities which might be called upon for normal postoperative care as well as for management of the unexpected.

My own child, for example, might have an elective herniorrhaphy carried out in one community under a given set of circumstances with my complete approval at the age of three weeks, in another at three months, and in a third I might not be completely happy with the risk at even three years. In the following discussion of the timing of certain elective pediatric operations the experience at The Children's Hospital of Philadelphia is drawn upon in large part. Similar advice might well be obtained from other pediatric surgical centers.

It is foolish to quibble about the proper timing of the repair of a cleft lip, for example, when one is talking about two weeks versus two months of age. In the given institution, if a team approach to repair of cleft lip has proved to be adequate at the age of two months, it would be foolhardy to push this well established routine into shifting to an earlier age when the confidence of any one member of the team might not be as great. On the other hand, for some elective surgical procedures, such as the repair of hypospadias, it is quite legitimate to argue in favor of preschool correction as opposed to a postpuberal correction. The pediatrician in such a circumstance might decide whether it is more important to have his patient's psychologic future protected by the completion of a series of operations before school resulting in normal urination or whether his surgeon's desire to work on a large phallus is worth the extreme psychologic trauma inflicted upon his patient by going through school sitting on a toilet to urinate like a female.

Obviously, good sense in the selection of the surgeon for a given job as well as satisfaction with the over-all care of the patient by previous experience will serve to make most decisions in surgical timing. The following is intended as a guidepost to strengthen or confirm these beliefs.

Hemangiomas

Hemangiomas must be classified according to specific type before one can make a definitive statement about therapy. In general, most surgeons agree that the port-wine stain is best left alone if not in a visible area of the patient's anatomy, or treated by cosmetic make-up such as Cover Mark if it is. A few clinics in this country are tattooing the purple areas. (See also p. 1018.)

For other hemangiomas, such as the capillary hemangioma, the hemangio-endothelioma, and so on, spontaneous remission is the rule and is to be expected. Such regression can, on occasion, be hastened by the use of judicious small doses of x-ray therapy, and to save psychologic trauma in a family situation, this is sometimes advisable.

Lesions on the face present to the surgeon and the family a problem that has yet to be solved. Two contrasting views have been put forward —early treatment to prevent spread, and the conservative plan permitting the lesion to run its course.[1, 5] At present in our clinic we are using both methods, giving very small doses of roentgen therapy early if the lesion is increasing in size, or following the course of others by observation. In contrast to 10 years ago, with accumulated experience we are becoming more and more conservative and using radiation less.

Nevi

Most patients have at least one nevus, and some have large numbers of them. Malignant change in these lesions is extremely rare in childhood, so that elective excision of nevi is carried out some time before puberty. If excision is decided upon, it should be complete, and electrodesiccation is not advised. Indications for removal include lesions that are deeply pigmented, whether hair-bearing or not, and those subject to constant trauma in belt, strap or collar lines. It is difficult to turn down a family's request for excision of a nevus based upon their fear of cancer. (See also p. 1021.)

Lymphangiomas

These are probably hamartomatous formations of the lymphatic system. They are usually discovered in the newborn period and should be excised without delay. The cervical region is the most frequent site, and some of these extend into the thorax. Less commonly lymphangiomas are found on the trunk or extremities. Every attempt should be made to dissect them out cleanly on the first occasion, since subsequent procedures become increasingly difficult. However, some lymphangiomas are so extensive that this is not possible. When the lesion involves the thorax from the neck, a procedure in two stages may be advisable.

Cleft Lip

The training and past experience of a surgeon are likely to dictate his choice of time for primary repair of a cleft lip. There has been a tendency of late to very early repair. As far as the lip is concerned, feeding is not a consideration in the timing. If the infant is in good condition, it is safe to do the repair in the first two weeks of life. Within this period there are additional advantages in operating in the first 48 hours of life. Should any doubts exist about the infant's general condition, correction should be delayed until weight gain has been established and the initial hemoglobin fall has shown an upturn. Technical preferences may guide the surgeon for the individual case so that repair can be advised between the limits of one day and 10 weeks. Some surgeons prefer to wait six weeks, believing that in this time any hidden anomalies or neonatal medical problems will have come to light.

Cleft Palate

Simple clefts not involving bone may be closed by six to eight months. The more usual complete cleft presents a much more controversial problem. Undoubtedly, the earlier cleft palate repair can be

effected ideally, the more normal will be speech development. The advantages of early surgical repair are offset by the vulnerability of the bone and developing teeth. In part, the decision must depend on a study of the general development of the child, a study of the facial bone growth and the presence of other anomalies. Deviations in any of these may warrant a delay in palatal repair. In the absence of delaying factors, the surgeon should aim to obtain closure of the palate by 18 to 24 months.

Malformed Ears

Congenital partial or complete absence of the anthelix results in prominent or protruding ears. Correction of this deformity in boys should be performed just before the child goes to school, about the age of five to six. This may avoid some of the torment meted out by other small boys. The necessity for treatment of the same deformity in females is lessened by the camouflage of long hair. Reconstruction of grossly deformed ears is an exceedingly difficult task, but when it is attempted, it should be done with a plan to complete the steps by school age. The personality of the child should play a part in the decision to operate. Some children seem impervious to the taunts of their fellows, and may well get along without corrective surgery. If surgery is to be undertaken after ridicule in school has forced the issue, a change of school after repair of the deformity is desirable if the operative correction is to be accompanied by the desired psychologic result.

Syndactyly

Although parents are anxious to have this deformity corrected as soon as possible, it should be delayed until the hand is large enough to bandage and splint safely. Skin grafts are often necessary, and scarring from inability to maintain dressings aggravates the problem. Undue delay, however, must be avoided, since stunting of one finger by the poorly developed mate may occur. If there is no stunting, the correction is delayed until three to five years, but it may have to be carried out earlier. Fusion of the tips of the fingers alone merits early separation by six months to allow undisturbed bone growth. Syndactyly of the toes seldom needs attention except in association with deformity and difficulty in fitting shoes.

Supernumerary Digits

Many of these are small skin tags that can be simply excised in the newborn period. The more complex supernumerary digits require

roentgen study to determine the anatomic arrangement of the bones, outlining the involved metacarpals or metatarsals as well as the phalanges. The optimum bone structure must be conserved for future growth and function. There is usually adequate skin covering available, and therefore early surgical correction is performed to encourage undistorted growth. In the absence of contraindications, this may be carried out at three months of age.

Branchiogenic Anomalies

Branchiogenic remnants (see also p. 1012) may appear as skin tags, skin tags filled with cartilage, cysts, sinus tracts, or a combination of several of these together. There is no urgency to correct these defects in the newborn period, and the age of election is dependent more upon the anatomic configuration of the child's neck than upon age. The short, fat neck of the infant is not conducive to easy dissection, nor does it provide the easiest patient for induction of anesthesia. Although some of these youngsters can be readily operated upon in early infancy, it seems prudent in others to wait until they have lost the chubby characteristics of the infantile neck by about the age of two years.

Other factors will dictate variations in the time of management. Large cartilaginous appendages on a neck sometimes produce sufficient emotional problems in parents of such children that early excision is mandatory. The question of the occasional infection of a sinus tract or a branchiogenic cyst may also force a surgeon's hand.

Thyroglossal Duct Remnants

A thyroglossal duct cyst (see also pp. 1011, 1025) with or without a sinus tract may arise at any point from the foramen caecum of the tongue to the suprasternal notch. The majority appear in the region of the hyoid bone or just below it, a cyst without a sinus being the more frequent lesion. All that has been said about branchial cysts could be repeated in reference to the thyroglossal duct cyst. It is desirable to excise the thyroglossal duct cyst before infection has taken place, since the latter circumstance frequently necessitates two operations instead of one and makes the second procedure more difficult than it might be if the operation could be undertaken in virginal territory. Failure to remove the central segment of the hyoid bone at the time of excision of the cyst and its duct invites recurrence.

Torticollis, or Tumefaction within the Sternocleidomastoid Muscle

The surgeon is asked to see patients suffering from the syndrome (see also p. 1015) ably discussed by Chandler[2] because of a tumor in the

lower end of the sternocleidomastoid muscle on one side, or because of facial and cranial asymmetry, or because of inability to turn the head equally to both sides. The mass in question is usually not discovered in the newborn nursery, but makes its appearance rather rapidly after discharge from the lying-in hospital. If presented to a physician at that time, the majority of patients can be managed conservatively with exercise, thereby eliminating the need for surgery and looking forward with considerable assurance to relief of immobility of the neck and facial asymmetry with the passage of time. The tumor itself, a fibrous affair of desmoid characteristics, disappears in time regardless of the therapy instituted.

Division of the sternocleidomastoid muscle on the affected side becomes necessary on rare occasions when the family cannot or will not carry out the exercises properly, or when, for some unusual circumstance, the exercises produce side effects such as respiratory obstruction.

Beyond the age of several months when the desmoid-like tumor has disappeared and is replaced by a thin, fibrous sternocleidomastoid muscle, division of the sternal and clavicular heads of the muscle is probably indicated in most cases if one is to obtain the best result for facial and cranial asymmetry.

It has been our custom to continue exercises for four to six weeks in those patients in whom the sternocleidomastoid muscle has been divided, but we have not resorted to the application of casts in any patient.

Hydrocele

A hydrocele (see also p. 1129) in an infant may well be a physiologic phenomenon which will result in disappearance of the swelling by about the age of three months. The presence of a hydrocele which fluctuates in size, grows in volume or is large and tense, practically always indicates the presence of a communicating patent tunica vaginalis or indirect inguinal hernia. Such a hydrocele then loses its indications for management, and resolves into the indications for care of the hernia.

Hydroceles should not be aspirated. There is the ever-present danger of infection, and aspiration of the physiologic hydrocele is unnecessary, and of the communicating hydrocele, futile.

Inguinal Hernia

In the absence of contraindications to an operation, an inguinal hernia diagnosed in the neonatal period should be repaired at the age of six weeks, but, as with other elective operations, much depends on available skillful anesthesia and surgical help. The procedure is usually

elective and can be timed in part for a family's convenience. Long delay, however, is unwise, since incarceration may occur at any time and complicates handling for surgeon and parents. (See also page 1129.)

A rare hernia causes trouble in the newborn period and should then be repaired without delay. But if the hernia is not symptomatic, it is an advantage to allow the infant to establish some weight gain and resistance and carry out the repair electively at six weeks.

Bilateral hernias can usually be repaired under one anesthesia, but if a patient is tolerating an anesthetic poorly, the surgeon should not hesitate to defer the second side to a later date rather than run any increased risk. Indications for delay beyond this period such as congenital heart disease or cystic fibrosis have to be considered carefully. The risk of possible incarceration has to be balanced against the risk of an operative procedure on the patient. Often indefinite delay is solved by attacks of incarceration forcing the surgeon's hand, and for that reason it is wise to plan elective repair if at all possible.

Umbilical Hernia

Most umbilical hernias (see also p. 1097) will close spontaneously, and we take the attitude that it is a rare one that does not. Strapping may perhaps increase the rate of closure, but seems to have too uncertain advantages to outweigh the disadvantages of possible excoriation and infection resulting from long-term adhesives on the skin with the inevitable soiling of the dressing.

Indications for surgical repair vary, and those of most surgeons will include (1) very thin atrophic umbilical scar; (2) incarceration of the sac contents—rare; (3) increasing size of defect; (4) persistence of defect without signs of improvement after the age of two to three years or beyond this age; (5) when the surgeon and family come to the decision that an operative procedure is desirable even in view of the fact that most umbilical hernias close spontaneously.

Undescended Testis

Conclusive evidence to determine the ideal age at which to perform orchiopexy is still lacking. It must rest upon a large statistical survey of those patients who have had bilateral undescended testes with one or both successfully brought down into the scrotum. Fertility studies based on offspring rather than sperm counts should yield the answer, but such a survey presents many problems. The Torek procedure should not be used, and the techniques in nearly all pediatric surgical services are probably comparable. Our studies have led us to believe that definite morphologic changes occur in the undescended testis

before the age of five years. For this reason orchiopexy is advised before that time. (See also p. 1137.)

The early limit is at present based on the technical problems encountered in working on a very small structure and the ensuing risks. However, in the younger child the structures are more pliant, there seems to be less need for much dissection of the cord, and the distances involved are smaller.

Under ideal conditions, then, orchiopexy is advised not before one year of age and not later than four years.

The unilateral undescended testis has less significance so far as fertility is concerned, but deserves to be corrected within the same time limits. There is still the associated hernia that requires repair, the value of a "spare tire," and the importance of correcting a physical defect before the child goes to school. Finally, the possible increased incidence of malignancy developing in an undescended testis cannot be ignored, difficult though it is to provide statistical evidence.

Hypospadias

Hypospadias varies in severity from a minimal failure of closure of the ventral groove of the glans penis to complete absence of fusion resulting in a urethral opening in the perineum. The former rarely requires attention unless the urethral opening is stenotic. The latter must be surgically corrected. Complete repair often includes releasing the chordee, enlarging of the urethral opening and reconstruction of the urethral canal on the ventral surface of the penis.

Several methods are advocated for this reconstruction. Whatever method is used, the following should be fulfilled. Reconstruction should be complete certainly before the boy goes to school. It should be associated with minimal psychic trauma, and late urethral stricture or fistula should be avoided. It is our aim to complete the hypospadias repair by the age of two years. Postoperative erections, which can be troublesome in the older child, rarely occur at this time. The child is able to micturate in the normal manner by the time he is concerned with keeping dry. The use of penile and preputial skin to reconstruct the urethra eliminates anxiety about future growth of the repaired area.

Epispadias

Epispadias is usually seen in association with exstrophy of the bladder, although it may occur rarely as an isolated lesion, or in combination with other major lower abdominal defects such as omphalocele or imperforate anus. Our effort, in general, is to accomplish the repair by the age of two years, but the indications for and demands of the other

surgical procedures associated with the epispadias may postpone such repair for several months to several years.

Exstrophy of the Bladder

To parents, exstrophy of the bladder must represent a terrifying problem, but in recent years this deformity has been re-evaluated, and reconstruction of the bladder in the newborn period may become the procedure of choice. We currently believe that most bladders should be turned in by about six months. If subsequent experience indicates that the result is less than desirable, one can then resort to ureteroenterostomy. The majority of our cases were treated in the past by transplanting the ureters into the sigmoid colon at six months of age. Care of the exposed bladder until this time consists in careful hygiene, using soap and water and frequent changes of diaper. If surface excoriation and bleeding are troublesome, application of sterile petroleum jelly to the diaper will solve these problems. Excision of the bladder and repair of the abdominal wall defect are deferred until the child is between 12 and 18 months old. Bowel training has not proved to be a problem and is complete by the age of three to four years.

Congenital Polyposis

A clear distinction must be made between multiple polyps of the large bowel and familial polyposis (see also p. 1158). The former responds to local excision of the individual lesions, though this may include large segments of the bowel, whereas familial polyposis carries the almost inevitable risk of future carcinomatous degeneration if the entire large bowel mucosa is not excised. The age at which malignancy develops cannot be definitely stated, but Gross[3] reports carcinoma with lymph node metastasis in one of his patients who was 12 years old.

Treatment of familial polyposis demands total colectomy with removal of all rectal mucosa, certainly before puberty and probably before 10 years of age. The technique we use is complete resection of the colon and upper rectum, leaving the lowest 2 inches of rectum intact. The mucosal lining of this short segment is then carefully and completely excised, and the terminal ileum is pulled through the remaining rectum. A few days later the ileal pull-through is trimmed and sutured to the anal margin. This perineal ileostomy is preferable to an abdominal ostium, and since some of the sphincter mechanism is preserved, these children can learn to control their bowels with little more training than a normal child. Abdominal ileostomy can always be resorted to later, if need be.

Splenectomy

The possible role of the spleen in preventing massive infection has been stressed of late. Although our experience does not support this concept, we have endeavored to delay splenectomy for hematologic disorders until after the age of two years.

Congenital Spherocytosis. Splenectomy for congenital spherocytosis is indicated when the patient shows signs that the hemolytic process is excessively active, resulting in a persistent anemia. If the spleen is not removed, the patient will be subject to sudden hemolytic crises and in addition will almost certainly have cholelithiasis in later life, a direct consequence of the exaggerated bilirubin production and destruction.

Thrombocytopenic Purpura. Owing to the beneficial supportive effects of adrenocortical hormones, emergency splenectomy is now rarely needed as a lifesaving measure in thrombocytopenic purpura. With these hormones it is usually possible to support a patient for six months without appreciable risk. If after this it is evident that the thrombocytopenic process is still active, with hypersplenic destruction rather than deficient platelet production, splenectomy should be performed. It will nearly always be followed by inhibition of the process.

Thalassemia Major. The spleen in thalassemia major grows to a great size and almost invariably becomes a mechanical problem. Hypersplenism may then be superimposed. Unless one's hand is forced by torsion or trauma, splenectomy is deferred until after the child is two years old.

Esophageal Varices

Bleeding from proved esophageal varices (see also p. 1155) is a danger signal that cannot be ignored. At whatever age it occurs, an investigation of the portal venous system is essential. Provided the spleen has not been removed, a percutaneous splenic pulp pressure and splenoportogram will provide pressure information as well as outline the venous channels. Liver function studies help to differentiate those patients with extrahepatic portal obstruction from those with cirrhosis.

If a good portal vein and splenic vein can be demonstrated, the opportunity exists for a splenorenal or portacaval type of shunt. One hesitates to do a splenorenal shunt in the small patient because the diameter of the anastomosis between the two veins increases the opportunity for thrombosis on the basis of size alone. Additional factors which should be considered comprise the associated hepatic disease with its concomitant findings such as ascites, hypoproteinemia, and jaundice which may alter the timing of operation.

When no adequate vein is found to shunt the portal circulation into the cava, we advise colon or jejunal interposition with resection of the upper half of the stomach and lower end of the esophagus. Our experience with the colon has been encouraging,[4] and it is, therefore, favored.

When the patient is bleeding profusely from varices, attempts are made to control this initially with transfusion and balloon tamponade. When control is achieved, the patient is kept in the hospital for 10 days to recover from the effects of the hemorrhage before a shunt is carried out. Unfortunately, at times there is no opportunity for this period of recovery and the operation has to be performed as an emergency.

Aganglionic Megacolon

The management of aganglionic megacolon (see also p. 1150) is rapidly becoming a problem of the neonatal period. A better understanding of this entity as a cause of intestinal obstruction in the newborn eliminates many of the problems in obstipation due to megacolon formerly seen in older children.

Most surgeons who have had experience with the management of aganglionic megacolon in the newborn period prefer a temporary colostomy in either the transverse or sigmoid colon followed by a definitive procedure when the youngster weighs 20 to 25 pounds. The definitive operation attempted in an infant is not only more difficult, but also carries with it a considerably increased risk. That it can be done is not denied, and in some parts of the world where culture will not permit the acceptance of an infant with a colostomy, definitive surgery is carried out in the newborn period (personal communication from J. Louw, University of Capetown, South Africa).

When youngsters with aganglionic megacolon have survived the neonatal period without proper diagnosis or surgery, their situation should be dealt with definitively as soon as possible. Whether or not preliminary colostomy is necessary in an individual patient will depend upon many circumstances which are not specifically a matter of timing.

Biliary Atresia

No liver function test at present can invariably distinguish biliary atresia from nonobstructive jaundice in the first few months of life (see also p. 1063). When apparent obstructive jaundice persists for eight weeks, a surgical solution should be sought. If the condition is amenable to correction, biliary drainage must be established before irreparable damage to liver function ensues.

Since major exploration is hazardous in the presence of hepatitis, the initial procedure at eight weeks is limited to a small transverse incision. Through this a catheter is introduced into the gallbladder for an immediate cholangiogram. If a normal biliary system is demonstrated, the catheter is tied in and the wound closed.

When normal ducts are not seen, the exploration is extended in the search for an anomaly which can be corrected. Unfortunately, this is rare.

Bronchiectasis

As a result of more effective medical prevention and management, bronchiectasis in childhood has been encountered with decreasing frequency as a surgical problem. There is no age of election for resection for bronchiectasis, and each case must be considered on the basis of the severity of the disease, the response to medical measures and the risks and advantages of surgical treatment. Pulmonary resection for this disease is seldom likely to be required in the first few years of life.[3]

Lung Cysts

A distinction must be made between congenital and acquired lung cysts (see also p. 1032). Congenital lung cysts may produce interference with normal respiration, and resection is an emergency procedure. If the cyst is discovered incidentally in the first few months of life, elective excision is advised before infection supervenes. In the older child the differentiation between congenital and acquired cysts is more difficult. A cyst appearing after a known pulmonary infection is likely to be a postinfectious pneumatocele and, if it is causing no respiratory difficulty, may be treated conservatively and could be expected to disappear. Signs of progressive enlargement or a fluid level indicating infection would justify surgical resection. (See also page 1043.)

Pectus Excavatum

Not only is the timing of the surgical correction for pectus excavatum a difficult decision to make, but in our opinion the selection of the patient is also not clear. There is no doubt that some infants with a funnel chest show spontaneous improvement as they grow, and there is ample evidence that many children who do show apparent cosmetic change in the configuration of the thoracic cage have no physiologic impairment when full grown. Our only warning about the timing of this procedure relates to the instability of the young child's thoracic cage when his costal cartilages are divided. For that reason we certainly

think operation should be postponed until beyond the age of three and are looking forward to more scientific methods of measuring cardiac and respiratory reserve so that there will be a firm indication for surgery in selected patients.

SUMMARY

The problems of surgery in the newborn cannot be covered in this discussion of timing in pediatric surgery. An attempt has been made to cover a number of the problems seen after this period and to outline some reasons and indications for the timing of surgical treatment. These patterns have been worked out over a number of years not only from our own surgical experience, but also from that of others in the field of pediatric surgery. It is worth pointing out that the other members of the team with special training—anesthetists, pediatricians, residents and nurses—play a vital role in enabling many of these patients to be handled safely at early ages. Without them, many of the times indicated would have to be modified.

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ANESTHESIA IN INFANTS AND SMALL CHILDREN

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Rapid advances during the last decade in the field of anesthesiology have greatly aided the development of pediatric surgery. The general principles of modern anesthesiology, which include preoperative evaluation, careful selection of agent and technique, close attention to adequate pulmonary ventilation and adequate circulation during operation and careful supervision of the patient in the immediate postoperative period, are the same for the adult as for the infant or child. For the infant or child the application of these principles must, however, be modified because of variations in size, anatomy, physiology, pathology and psychology.

The aim of the anesthesiologist is to provide the most pleasant sleep for the patient, and the optimum working conditions for the surgeon with the minimum of risk of mortality or morbidity. I am often asked about the risk of anesthesia in children as compared with the risk in adults, or about the risk of anesthesia within the first six months of life as compared with the risk thereafter. The questions are pertinent, especially when the surgeon has to select the optimum time for repair of hernia or hypospadias, operations which are not urgent, but which should be done as soon as possible. Convincing data of this are elusive. The data available indicate that the risk of anesthesia in the first decade of life is greater than in any other decade except the ninth or tenth.[2] These data were collected in 10 large university hospitals over a 5-year period during which pediatric anesthesia was in its infancy and in locations in which pediatric anesthesia was not especially emphasized. I believe that if proper emphasis is given to the special problems of pediatric anesthesia, the risk of anesthesia should be no greater in the first decade than in any other decade, no greater in the infant than in the older child.

PREOPERATIVE EVALUATION AND SEDATION

The preoperative preparation of the child for anesthesia and surgery begins in the referring physician's office (see also p. 979). The child and his parents must be prepared psychologically to face the experience of hospitalization. They should be reassured that modern methods of anesthesia can provide the child both safe and pleasant sleep for his needed operation. The referring physician should not neglect the physical preparation. The child is especially prone to sudden respiratory infections during the winter months. Operation should be deferred until the child is free from these infections. Simple physical examination and laboratory survey will rule out heart murmur, anemia and renal disease, conditions which must be investigated and treated before the optimum anesthesia results can be achieved.

Once the child has been admitted to the hospital, the anesthesiologist should visit him to survey the physical status in order to determine the best agent for this child. He should assess the psychologic make-up of the child to determine the selection and dose of preoperative drugs. The use of preoperative drugs is extremely important in children. It has been shown that children who have terrifying experiences in the operating room can develop disabling psychologic symptoms such as night terrors, enuresis and temper tantrums.[8, 10] Fortunately, there is available a wide variety of drugs which can provide sedation in children.* The drug should allow the child to be transported to the operating room moderately asleep; it should allow induction of anesthesia with the child neither recalling the trip to the operating room nor awakening during the induction. In selecting the proper medication the anesthesiologist should balance these aims against the physical status of the child, the choice of the anesthetic agent and technique,† the skill of the anesthesiologist, and the facilities available for the preoperative and postoperative surveillance of the patient.

At The Children's Hospital of Philadelphia 290 patients were studied and 12 combinations of drugs were used in order to evaluate the most effective means of sedation for children.[9] We have found that combinations of pentobarbital, morphine and scopolamine best meet the aims of preoperative sedation in the child over 15 pounds who is in excellent physical condition. In the infant under 6 months or 15 pounds no sedative drug or narcotic is used, and atropine is substituted for scopolamine. Other workers have found combinations that seem to fit the conditions of their practice better.[14] I must point out that few

* Pentobarbital, morphine, Demerol, Vesprin, promethazine, thiopental.

† Certain premedications depress respirations; the technique of "open drop ether" does not allow the anesthesiologist to assist respiration. This method of induction can, under these circumstances, be prolonged and difficult.

well controlled studies of the effectiveness of these drugs have been done, and it is wise to maintain a critical eye upon the drugs suggested pending better evaluation.

CHOICE OF AGENTS AND TECHNIQUES

The child then arrives in the operating room prepared, evaluated, and asleep, ready for induction of anesthesia. Generally speaking, children dislike intravenous needles. Inhalation methods of inducing anesthesia are better tolerated and more easily applicable. The gas can be gently blown over the face or presented through a toy if the child is still awake. Cyclopropane is the best agent for accomplishing this induction, but nitrous oxide, trichloroethylene vapor, halothane vapor and Vinethene vapor have been used with success.

Once anesthesia is produced with an inducing gas, the conduct can proceed in many directions. *Ether* is used most commonly as the maintenance anesthetic on healthy children during operation. There is real reason behind the popularity of ether in children despite its decreasing use in adults. Brewster and Isaacs[6] showed that ether is a cardiocirculatory depressant in the absence of epinephrine and norepinephrine in dogs. It seems clear that many of the cardiac problems previously found in elderly patients undergoing ether anesthesia were due to this factor. Price[11] has shown that in man there is an increased circulation of norepinephrine during ether anesthesia. However, Brewster, Bunker et al.[7] have shown that ether produces a metabolic pattern in children not unlike epinephrine stimulation. Perhaps, then, the child behaving more like a dog than adult man protects himself against the depressant effect of ether by secreting epinephrine. In addition, the child's circulatory system is usually more reactive and healthy to begin with than that of the average adult undergoing surgery. Another dividend of ether anesthesia is the fact that ether does not cause respiratory depression except in the deepest levels of anesthesia, so that assisted or controlled respirations are not necessary. Ether, then, is an excellent anesthetic for maintenance in healthy children undergoing surface operations.

If the child is debilitated or in circulatory failure, or requires a prolonged operation in the chest or abdomen, ether loses most of its advantages. In these instances *cyclopropane* is the agent of choice since it rarely causes circulatory collapse, provides excellent relaxation and a quiet mediastinum in relatively light planes of anesthesia. In addition, prolonged anesthesia and operation do not mean prolonged recovery periods. With cyclopropane the anesthesiologist must be skilled in maintaining adequate ventilation; in children this is best accomplished by rapid controlled respiration.

All the agents and techniques used in adults have been tried and even advocated for use in children. There are many which have proved useful. Trichloroethylene can be used successfully as a nonpotent anesthetic. Halothane is a new nonflammable, potent anesthetic vapor. Regulation of dosage with thiopental, the major intravenous agent, is difficult in small children.

Local or regional techniques have little real application in children. Occasionally a situation develops when such a technique is the procedure of choice. It has been demonstrated that brachial block, spinal anesthesia and epidural anesthesia can be accomplished successfully and safely in children.[13, 14, 15]

In our opinion the use of muscle relaxant drugs is valuable in pediatric anesthesia and holds a definite place in the management of certain situations. The use of these drugs presumes the use of an endotracheal tube and the most vigorous support of ventilation.[17] Stead[18] has demonstrated that the small infant is more sensitive to d-tubocurare and less sensitive to succinylcholine. Infants and children tolerate the latter well. Muscle relaxants are usually administered intravenously, but succinylcholine has been shown to be effective intramuscularly in children. In our experience we have not encountered prolonged postoperative apnea or circulatory collapse in over 903 administrations of muscle relaxants in children.

The use of the endotracheal tube in infants and children has undoubtedly introduced a safety factor. In 10 years over 20,000 patients at Children's Hospital of Philadelphia have been subjected to this maneuver with only minimal complications. Certainly great care must be taken to ensure gentleness of manipulation and scrupulous cleanliness of instruments.[16]

In a careful study of 1200 patients subjected to endotracheal intubation 56 were found to have had some respiratory complication varying from hoarseness to stridor.[1] None required tracheotomy. The factors which were important in the children developing croup were (1) age—in the one- to three-year age group; (2) sex—larger proportion were males; (3) length of operation—the longer the operation, the greater risk of complication; (4) season of the year—a large number occurred in the Fall. None of these measured factors, however, was important enough to deny a child the use of the endotracheal tube.

RESPIRATORY FUNCTION DURING ANESTHESIA

Of the vital systems, the respiratory system of the infant is the least adaptable to the hazards of anesthesia.[12] Even though the respiratory limitations decrease as the child grows older, the relative vulnerability of the respiratory system remains until adult life. Constant attention to this system is mandatory throughout the anesthetic period. The

airway must be maintained and ventilation constantly assisted or controlled as the situation might require. In the small infant diaphragmatic respiration is predominant. It is important to minimize any occurrence during anesthetic management that limits diaphragmatic function, such as abdominal distention, heavy drapes, Trendelenburg position, and surgical assistants' elbows.

Research into the regulation of ventilation of newborns has not yet given us a clear picture of this aspect of physiology. Certain facts arising from the study of physiology are of practical value in anesthesia. Irregular rhythms of respiration are made more regular with oxygen. Regular rhythms are made irregular by hypoxia. Increase of inhaled oxygen increases minute volume; decrease of inhaled oxygen decreases minute volume. It is not desirable in infants to allow hypoxia or hypercarbia to accumulate in order to stimulate respiration.

CIRCULATORY FUNCTION DURING ANESTHESIA

Fortunately the circulatory system of the infant or small child can tolerate the ordinary stresses of anesthesia better than the respiratory system. Usually the heart is strong, the vessels are patent and supple.

However, although the circulatory system can withstand the ordinary stresses of the anesthetic state, the compensatory ability of the infant circulatory system is low. For example, blood loss can be especially insidious, since the only warning of impending danger will be sudden complete circulatory collapse. Ordinarily the infant has a rapid pulse. Maintenance of the pulse and its amplitude are the most reliable signs of the infant's general condition during anesthesia. The response to most noxious stimuli will be bradycardia. The bradycardia itself leads to circulatory anoxia, and a vicious cycle is instituted. Blood replacement in infants requires particular attention. It must be always remembered that what would seem to be a small blood loss in an adult might be an appreciable fraction of the blood volume in the infant.

HYPOTHERMIA IN PEDIATRIC ANESTHESIA

In 1951 Bigler and McQuiston[4] demonstrated that under the conditions of their operating rooms, children from 1 to 13 years of age exhibited a progressive rise of body temperature. They believed this hyperthermia to be hazardous because it led particularly to convulsions, and they recommended cooling these youngsters. Later, hypothermia as a method of reducing oxygen utilization in occlusive heart surgery became popular.[19] Induced hypothermia has by now been applied to a wide variety of major operative procedures. The rationale for its use is that decreases in oxygen requirement with each degree drop in temperature provide a wider margin of safety in situations in which one or more

vital tissues might be deprived of oxygen for periods of time. Although hypothermia itself causes physiologic derangement, its use appears to be valuable and well accepted both in adults and children in (1) occlusive cardiac surgery, provided the occlusion is no more than seven minutes; (2) large neurosurgical operations; (3) massive abdominal dissection of liver, aorta, kidney. In addition, Brayton and Lewis[5] demonstrated that the techniques and information gained in so-called deep hypothermia (cooling from normal to approximately 30° C. or 88° F. after induction of anesthesia) could be applied to the hyperthermic child who requires emergency surgery. They performed mild hypothermia (cooling to normal temperature or slightly below) in a series of febrile children in preparation for surgery and demonstrated that the use of this technique decreased the hazards of convulsions, coma and circulatory collapse. Our experience with the use of mild hypothermia in the febrile child completely confirms the observations of Brayton and Lewis.

MONITORING IN PEDIATRIC ANESTHESIA

Another concept in anesthesiology that has had particular application in children is "monitoring." "Monitoring" is defined in this instance as something that serves to remind or give warning. Therefore monitoring includes all the traditional parameters followed by the conscientious anesthesiologist, such as pulse, auscultatory heart beat, blood pressure, skin color, skin turgor and, in addition, but not superseding these methods, other physiologic data which can be presented electrically. It is now practical to record during anesthesia the electrocardiogram, electroencephalogram, the direct arterial blood pressure, blood oximetry and carbon dioxide content of alveolar air. The information thus gained can be displayed upon an oscilloscope or upon a sheet of paper by pens, or even translated into sound. In anesthetizing children, the student with prior experience in adults is struck by the capriciousness of changes in the infants' respiration, depth of anesthesia, blood pressure, pulse and general condition. He is impressed that the child requires much closer watching in order to ensure an anesthesia free from complications. The advent of the newer methods, coupled with careful observations of the traditional techniques, has given the pediatric anesthesiologist powerful tools to provide a smooth and safe anesthesia.

RECOVERY PERIOD

Careful observation and management of the infant and small child in the immediate recovery period are vital to the successful conclusion

of the surgical procedure. In children, as with adults, the provision of a special nursing unit for the recovery of the patient close by the operating room suite has been found extremely useful. There trained personnel with drugs and equipment immediately available can observe and treat the sudden respiratory obstruction or circulatory collapse which can befall the patient after operation.

SUMMARY

It has been demonstrated that anesthesia can be successfully accomplished for the most major surgery in small premature infants to larger, older children.

I believe that anesthesia results in this age group can be achieved comparable to those in any age group if the following axioms are applied to the unique anesthetic problems of infants and small children: (1) careful preoperative evaluation and sedation; (2) rational selection of agents and techniques of anesthesia; (3) provision of a patent airway with the endotracheal tube; (4) constant support of ventilation during anesthesia; (5) quantitative replacement of blood loss; (6) conscientious monitoring of the patient's condition at all times; and (7) careful observation of the patient in the immediate postoperative period.

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VISIBLE LESIONS OF THE NECK IN CHILDREN

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From birth to 15 years of age children display a wide variety of visible lesions of the neck. Some are tiny, such as a brown nevus a centimeter in diameter; others are enormous, such as a cystic hygroma. The lesions range from cystic to solid and from benign to malignant. Some become inflamed; others hemorrhage. A small number must be treated urgently if the child is to survive. Many may be excised at a time of election to prevent penetration, invasion, infection or recurrence.

This presentation aims to indicate the spectrum of the pathology and to highlight some features of the diagnosis, management and prognosis.

MALFORMATIONS

Fetal development of the neck and surrounding structures is complex. Architectural anomalies constitute a wide variety of disorders, many of which manifest themselves as visible lesions in early infancy as well as in late childhood. Grooves, cystic bumps, appendages and sinuses are best understood in the complex framework of the embryology of the primitive branchial arches. Most of these embryonic defects require operation at an appropriate age and state of development.

Thyroglossal Duct Cysts and Sinuses

Swellings in the midline or close to the midline of the neck, between the apex of the mandible and the hyoid bone, are thyroglossal cysts

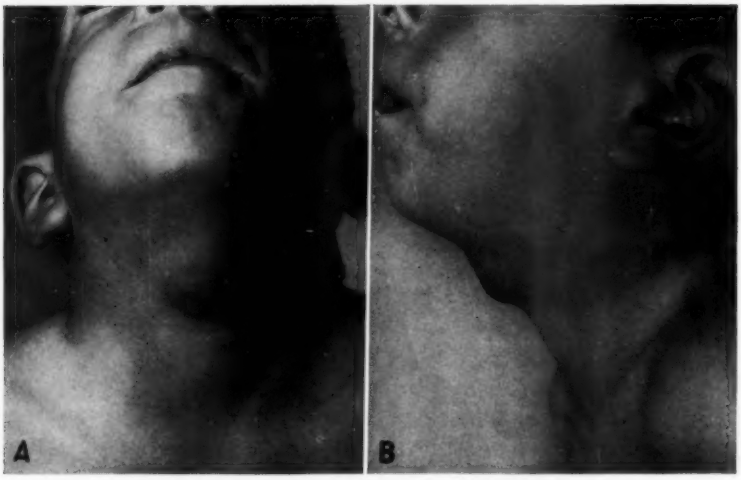

Fig. 14. Front and lateral views of thyroglossal duct cyst.

until proved otherwise (Fig. 14). These malformations persist after the thyroid anlage has migrated from its point of origin. A sinus connects the foramen caecum at the base of the tongue and the inframandibular cystic swelling, thus permitting intermittent accumulation of pharyngeal secretions. The sinus tract always traverses under the hyoid bone.

Once a thyroglossal cyst becomes filled with secretions, every effort should be made to prevent internal infection. If infection first inflates and identifies the lesion, all efforts should be bent towards control without incision and drainage. Surgical decompression is done only for abscess formation.

As soon as the child's neck has slenderized—any time after the second birthday—and when the visible midline swelling is in an uninflamed phase, the thyroglossal cyst together with its sinus, including a wedge of hyoid bone, must be removed all the way to the base of the foramen caecum. Superficial excision of the cystic portion is incomplete surgery and invites recurrence.

Branchiogenic Cysts and Sinuses

The first branchial cleft gives rise to preauricular swellings and fistulas, but persistence of the third, fourth and fifth branchial clefts results in cystic swellings or mucus-draining fistulas along the anterior border of the sternocleidomastoid muscle. These may be unilateral or bilateral. They may be infected or uninfected. While bathing the infant, the

mother may detect one as a pinhead-sized indentation of the skin of the neck. Sometimes a mucus discharge is observed. Occasionally throat infections activate and direct attention to a pre-existing branchiogenic cyst or sinus at any age in the pediatric range.

Surgical excision of the entire anomalous tract from cervical skin to the pharyngeal wall attachment is mandatory. The optimum time for operation is usually after the second birthday. If infection occurs, surgical dissection is ordinarily deferred until the inflamation has been quiet for at least six weeks. Cannulation with a stiff nylon suture, a horsehair or a malleable silver probe facilitates the dissection. Injection of the tract with methylene blue at operation is often messy, confusing and disappointing.

Ectopic Cervical Swellings

Visible swellings on either side of the neck or in the midline may be ectopic islands of tissue from the thyroid gland or the thymus. These are often firm and ovoid, and devoid of infection. If they are observed for a number of weeks, there is no particular change in size or consistency. Eventually such ectopia are surgically excised for cosmetic reasons and have no sequelae. In older life the aberrant thyroid takes on increasing significance as a primary site of malignant growth. Such lesions should be removed in early childhood after ascertaining that a normal thyroid is present.

The cartilaginous components of the branchial arches also persist as ectopia, both in the midline and on the sides (Fig. 15). Their removal is primarily of cosmetic significance. Some are removed in infancy, but larger lesions are excised when the neck slenderizes around the second birthday.

Fig. 15. Ectopic skin and cartilage.

Fig. 16. Bilateral web neck after correction of the right side by Z-plasty.

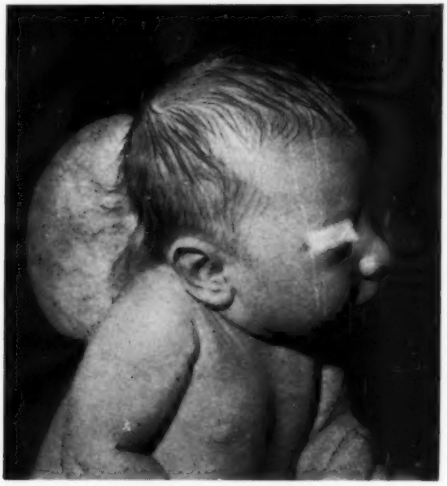

Fig. 17. Cervical meningocele.

Web Neck

Pterygium colli, or web neck, manifests itself unilaterally or bilaterally as a tough, thick fibrous web of tissue extending from around the ear out to or towards the shoulder cap (Fig. 16). The cause of the lesion is unknown. Associated cervical ribs do occur as well as an occasional malpositioned scapula of the Klippel-Feil syndrome. A few girls have

also had associated ovarian agenesis. The treatment is purely cosmetic and often is done in the preschool period around five years of age or even later. Multiple Z-plasties dispose of the web, but abnormal areas of hirsutism sometimes need to be disposed of by electrolysis.

Cervical Meningocele

Failure of the neural canal to close completely over the lumbar zone is not uncommon. Cervical meningoceles, on the other hand, are rather rare. Such cervical swellings are removed in the first few days of life. Often the pedicled base is reassuringly small and simple to close. A few, however, require laminectomy and careful occipital decompression to avoid subsequent neural canal obstruction (Fig. 17).

INFANTILE CORTICAL HYPEROSTOSIS

Attention is directed to the infant or small child with a visible swelling close to the mandible or clavicle simulating chronic cervical adenitis or osteomyelitis. Caffey has well documented the cardinal symptoms and signs of infantile cortical hyperostosis, i.e., irritability, fever, leukocytosis, pain and tenderness as well as visible and palpable swellings in the neck. The x-ray findings are diagnostic. Such patients should not have operation, and supportive treatment uniformly results in improvement over a considerable period of time.

WRYNECK

A not uncommon visible lesion on the neck of infants is torticollis. Often while bathing the baby, the mother detects a lump ranging from 1.5 to 5.0 cm. in diameter on the side of the neck in the middle or lower third of the sternocleidomastoid muscle. After a visit to her physician it is called to everyone's attention that the child's head is tilted to the affected side, that the chin can turn to the contralateral shoulder, but the baby resists turning to the ipsilateral side. Occasionally torticollis is noted by a neighbor or visiting grandmother who comments on the asymmetrical cheek bones or eyes. Often the back of the head is flattened on the dependent side.

Fremont A. Chandler attributed the lesion to intrauterine malpositioning causing undue pressure and altered vascular dynamics in the sternocleidomastoid muscle; 35 to 50 per cent of such babies arrived by breech. Whatever the cause, a firm, immobile mass is usually palpable in the affected muscle. This slowly enlarges from birth to six weeks of age. Occasionally it disappears, but most often it predisposes to muscle shortening, asymmetrical facial development and scoliosis.

The key to successful treatment is early vigorous massage and stretching exercises. If the mass is large, rock-hard and discrete, and has not responded to systematic stretching and massage, surgical excision is simple, safe and curative. Careful attention must be directed to the deep carotid sheath and the spinal accessory nerve. The former is behind the deep muscle belly, and the latter emerges from behind this superficial muscle in the upper third of its course. The fibrohematoma in the muscle causing the wryneck may involve the deep muscular component or the superficial, or both. Only involved muscular tumor needs excision. The prognosis with early detection and prompt vigorous management is uniformly satisfactory.

CERVICAL ADENITIS

As long as children have upper respiratory tract infections such as tonsillitis and pharyngitis, some of them will have acute cervical lymphadenitis (Fig. 18).

Fig. 18. Cervical adenitis.

It is well to remember that a 10- to 14-day course of a single antibiotic or sulfonamide drug is much more likely to cure acute or subacute infection of cervical lymph nodes than 2 to 3 days on each of half a dozen different drugs. The more prolonged exposure to a single compound is also less likely to produce strains of organisms resistant to our entire armamentarium. As in infections elsewhere in the body,

healing is favored by rest of the affected part and heat in the form of warm moist compresses.

If all conservative measures fail and fluctuation is present, incision and drainage will hasten healing. This is best performed by incising only the skin and continuing the dissection with a blunt instrument, since sharp dissection endangers the mandibular branch of the facial nerve and may cause permanent paralysis of the ipsilateral corner of the mouth.

If no obvious primary source of infection is found, the patient should be thoroughly evaluated for the possibility of cervical tuberculosis. Skin testing and a chest x-ray are frequently helpful, but usually a positive diagnosis is established only after histologic examination.

Surgical intervention in tuberculous adenitis is usually reserved for this diagnostic role or for the treatment of far advanced liquefied or secondarily infected nodes.

Several recent investigations have shown that the cervical tuberculosis we have all been seeing with increasing frequency for the past two to three years is due to a chromogenic variant of Mycobacterium and is no longer caused by the bovine or avian strain of *M. tuberculosis*. Fortunately these chromogenic organisms usually respond well to antimicrobial therapy with streptomycin, isoniazid and PAS. Even the presence of a draining sinus, spontaneous or surgically caused, need not cause despair, since most respond rather quickly to a well planned regimen of these agents.

TUMORS

Cystic Hygroma

Lymph-filled cystic hygromas (see also p. 1026) are not uncommon visible lesions of the necks of children in the supraclavicular fossa. Occasionally they are present at birth; indeed they may be so large that they impair delivery (Fig. 19, *A*). At times they are small in infancy and grow with the development of the child (Fig. 19, *B*), and at other times they suddenly appear in older children with attending infections in the throat, the teeth or the ears. Most are multicystic, and the lobules range from a few millimeters to several centimeters in diameter. A few seem small, but are like icebergs with only 10 per cent above the surface. Deep extensions may involve the subscapular area, the mediastinum, the trachea, the tongue and the carotid sheath.

Although cystic hygromas are essentially a tumefactious malformation, they can be as lethal as any metastasizing cancer by virtue of possible local extension, hemorrhage, or infection. They never respond satisfactorily to x-ray or radium therapy. The injection of sclerosing agents is hazardous, since they may be swept into the general circula-

Fig. 19. *A*, Large cystic hygroma. *B*, Cystic hygroma in a year-old child.

tion. Aspirations are rarely permanently beneficial, but may be life-saving when there is tracheal compression. Aspiration may be dangerous because of ensuing hemorrhage into the spaces previously filled with lymph.

The treatment of choice is local excision under general anesthesia whenever the diagnosis is suspected. To remove 2 dozen lymph locules at 2 weeks is often easier than trying to remove 2000 locules at 2 years of age. Operation is recommended at as early an age as possible. In the presence of an extensive cystic hygroma it is sometimes judicious to leave part of the tumor behind rather than kill the patient with heroic surgery. Temporary tracheotomy may be necessary. Preoperative chest x-rays are mandatory, since 10 to 15 per cent of cervical cystic hygromas have intrathoracic extension.

Hemangioma

Superficial capillary *strawberry hemangiomas* often appear as tiny dots on the necks of small babies. These visible lesions tend to enlarge with the infant's growth for about 6 to 12 months. Most stop enlarging by 18 months of age; thereafter spontaneous involution proceeds centripitally. Unless there is abnormally accelerated growth or severe bleeding, conservative management eventually is rewarded by silver-gray involution and disappearance of the lesions.

Cavernous hemangiomas, on the other hand, are inclined to develop large cavernous sinusoids with radial nutrient vessels. These thicken their endothelium a little with maximum irradiation, but most often need surgical excision.

Hemangioendotheliomas have small capillaries in a dense stroma, never fully empty under compression, look extremely malignant microscopically, but are only locally difficult to excise in toto. Minute seedlings tend to recur, and x-ray therapy is of little avail (Fig. 20).

Carotid Body Tumor

This variety of tumor on the side of the neck is rare in adults. In children it is rarer still, but does occur and sometimes becomes malignant. Early removal is mandatory to permit the surgical dissection from the carotid vessels without sacrifice of the internal carotid artery and attending cerebral thrombosis.

Tumor of the Thyroid

Numerous recent reports have directed attention to the increasing incidence of carcinoma of the thyroid in children (see also p. 1208). The absolute evidence to correlate previous exposure to x-ray irradiation with malignancy of the thyroid is still lacking. None the less, visi-

Fig. 20. Hemangioendothelioma in a 2-month-old infant.

Fig. 21. Thyroid tumor (Hurthle cell type) in a 10-year-old girl.

Fig. 22. Rhabdomyosarcoma.

ble lumps in the thyroid of children demand operation. Most of these lumps are followed up conservatively for a short interval; but after failure to disappear on iodized salt and the exclusion of cretinism, the persistence or enlargement of such thyroid swellings demands surgical excision. About 15 to 20 per cent of present day thyroid adenomas in children are malignant. On the other hand, Hurthle cell tumors (Fig. 21) and thyroiditis are also encountered.

Sarcoma

Though uncommon, rhabdomyosarcoma may present a visible swelling on a child's neck (Fig. 22); so may osteogenic sarcoma and fibrosarcoma of the mandible. Even with radical neck dissection, the removal of rhabdomyosarcoma is seldom curative. On the other hand, hemimandibulectomies for the removal of sarcomas of the jaw in children may be followed by excellent survivals. Lymphosarcoma is better treated medically than surgically, and a number of patients have had encouraging remissions.

Miscellaneous

Lipomas, neurofibromas, epidermoid inclusion cysts, calcified wens, sclerosed hematomas, localized lipofibrolymphangiomas—all these conditions cause visible lesions on the neck. All are benign. All are eventually removed after an interval of conservative watchful waiting.

NEVI

Nevi occur on the neck of children rather frequently. Their color ranges from none through pale tan to dark brown or black. Some gradually darken with age. Not a few become hirsute; occasionally the surface roughens with a verrucal pebbly appearance. All lesions which will subsequently be subject to continuous irritation, as under a boy's shirt collar, should be removed in childhood. Any nevus with malignant potentiality may misbehave after adolescence and is best removed before the twelfth birthday. Many times nevi on the neck are observed through infancy and early childhood when they are tiny and easy to remove; this is the age of election for surgical removal rather than later on when complicated Z-plasty, advancing flaps or grafts are necessary to fill the defect after removal (Fig. 23). Port-wine nevi are almost impossible to remove; yet cosmetic tattooing, though tedious, may help to cover the discolored area. The use of makeup may be the simplest solution in older children.

Fig. 23. Pigmented nevus which required removal and closure by plastic surgical methods.

CONCLUSION

1. Visible lesions of the neck in children constitute a common problem throughout the pediatric age span.

2. The basic pathology includes malformations, metabolic disturbances, infections and tumors.

3. A kaleidoscopic review is presented in an effort to highlight diagnosis, clinical management and prognosis.

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RESPIRATORY OBSTRUCTIONS OF SURGICAL IMPORT

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Few diseases afflicting infants and small children are as frightening and dangerous as those producing respiratory obstruction. Few conditions are as demanding of the utmost in care and skill in producing a satisfactory result. Furthermore, it is almost always an urgent situation leaving little time for delay in diagnosis and treatment. It is our purpose in this paper to review the various causes of respiratory obstruction of surgical import in infants and children, with emphasis on methods of recognition and diagnosis and the general principles of treatment.

RESPIRATION IN THE NEWBORN

Before studying the obstructive phenomena, it is valuable to consider some aspects of normal respiration peculiar to this age group. In the newborn a state of respiratory insufficiency is present because the lung alveoli are not immediately fully developed. While this development is taking place the infant's respiratory reserve may be precarious. Respiration is carried out almost entirely by the diaphragm, while the thoracic wall acts more or less as a fixed point from which the diaphragm can work.[9] Because of the weakness of the cartilages and ribs in the newborn there is a certain amount of paradoxical respiration that diminishes the efficiency of respiration. This is particularly true in the premature infant. The newborn with a limited tidal volume of 15 to 20 ml. will respond to a respiratory stimulus by increasing his rate, already normally rapid, rather than by increasing his depth of respiration. In this instance an increase in rate is not an efficient response. The infant's mediastinum is not so fixed as that of the adult, and it shifts alarmingly under certain pathologic circumstances. The trachea and bronchi have lumens of exceedingly small caliber and hence are easily blocked, from without as well as from within. Thick bronchial secretions and aspiration of regurgitated material are considerable hazards. Laryngeal edema may occur readily when the larynx is irritated. The cough mechanism is poorly developed in the infant.

In observing a newborn with respiratory distress one should determine whether there

is just dyspnea or just cyanosis, or both.[11] If cyanosis continues after preliminary resuscitation and the infant has little dyspnea, one is likely to have either central nervous system involvement with hemorrhage, or a congenital heart disease. If the newborn has both dyspnea and cyanosis, one should observe the respiratory rate and the type. Infants whose respiratory rates show a significant increase during the first few hours and days after birth have a high rate of morbidity and mortality. If the respirations are stertorous and the infant has a "mashed-potato" cry, most likely there is an obstruction in the pharynx. Laryngeal obstruction is characterized by rapid labored breathing, inspiratory stridor and an abnormal or absent cry. Rapid respirations with supraclavicular and intercostal retraction and with a normal cry usually indicate an obstruction below the larynx.

Because of rapid respirations and movement, roentgenograms must have a short exposure time and relatively high milliamperage. Unless special bedside units with these capabilities are available, such studies should be properly made in the radiology department. Films are then much more likely to be satisfactory for proper evaluation and diagnosis.

NASAL OBSTRUCTION

Anterior *choanal atresia* is recognized on sight. Posterior choanal atresia presents serious difficulty only when it is bilateral, or in the unilateral instances when the normal side becomes obstructed with infection or mucus. The condition should be suspected with intermittent respiratory distress, particularly during feeding, because the infant cannot breathe and eat simultaneously through the same orifice. One such infant was observed with continuous respiratory distress and a near-fatal outcome because instinct did not prompt him to open his mouth to breathe.

The diagnosis is established by inability to pass a metal probe or flexible catheter into the nasopharynx or by instillation of radiopaque medium into the nasal passages. Expiratory ballooning of the hypopharynx has also been described as a diagnostic radiologic sign.[16] (See also p. 956.)

In most cases the obstruction is bony. Operative perforation of the membrane or bony atresia with splinting by plastic tube for several days will effect a cure.[10, 13] Tracheotomy is often necessary, and tube feeding is frequently required.

Nasal encephalocele (rhinoencephalocele) and *pharyngeal tumors* such as neurofibroma may produce nasal obstruction in infancy. Because of the dangers complicating inadvertent opening of such a meningocele, roentgenograms of the base of the skull should be studied before any nasopharyngeal mass is attacked surgically.

PHARYNGEAL OBSTRUCTION

Macroglossia in infants is usually a relative enlargement of the tongue, which is usually outgrown. A true hypertrophy of the tongue is rare. Sometimes vascular bed lesions such as hemangioma or lymphangioma

are responsible for considerable enlargement. These may be either localized or diffusely spread throughout the tongue. Any of these enlargements may obstruct the air passages, producing distress.

In instances of mild obstruction treatment consists in maintaining the infant in a face-down position. Partial glossectomy is sometimes indicated for true muscular hypertrophy of severe degree, or in those instances in which there is no improvement as the child grows larger. Definitive treatment of the vascular lesions depends upon localization and type, and consists in either surgical excision, injection with sclerosing agents, or irradiation. Occasionally tracheotomy must be instituted before any definitive treatment can be done.

Hypoplasia of the mandible, or micrognathia (see also p. 957), is due to failure of the mandibular ridge to make contact with the maxillary ridge. This results in a relative macroglossia and glossoptosis, which may produce respiratory distress and cyanosis in the newborn. When associated with cleft palate, the condition is known as the *Pierre Robin syndrome*. Subluxation of the mandible will resemble this condition and may be ruled out by roentgenograms.

Treatment of the milder forms consists in keeping the infant in the face-down position. More severe forms are sometimes treated by suturing the tongue forward to the lower lip for several months until the mandible becomes relatively larger. This suture may cut through the tongue, however, and obstruction recur suddenly during a period when the infant is unobserved. A variety of devices exerting traction on the mandible have been devised, but results have not been satisfactory. Despite the difficulties encountered with tracheotomy in the newborn, it seems that this is the safest and most satisfactory temporizing procedure, at least in the severe cases.

Thyroglossal duct cyst (see also p. 1011) may be seen at the base of the tongue and, when large enough, may interfere with inspiration. Treatment is by excision. In an emergency, aspiration of the cyst may be lifesaving. A solid tumor in the same position may be a *lingual thyroid*, and it may be all the thyroid tissue this patient has. This status can be accurately determined by radio-iodine studies. Complete excision followed by appropriate thyroid replacement therapy is recommended as the treatment of choice.[15] It may be profitable to implant some of the thyroid tissue into the sternocleidomastoid or rectus muscle.

Ranula is the term applied to a retention cyst of the salivary glands in the floor of the mouth. As the cyst enlarges, it pushes the tongue upward and backward, occasionally producing respiratory distress. Treatment consists in surgical excision of the cyst if at all possible; or at least the cyst should be unroofed. *Dermoid cysts* and *cystic hygroma* (see below) of the floor of the mouth[14] may produce the same situation.

EXTRINSIC OBSTRUCTION IN THE NECK AND MEDIASTINUM

Cystic hygroma (see also p. 1017) is a congenital cystic tumor most frequently arising in the neck. The most credible theory of the origin and pathogenesis of this tumor has been proposed by Goetsch,[4] who stated that it probably arises from sequestrations of the lymphatic tissues derived from the primitive jugular sacs, which have failed to join the lymphatic system in the normal manner. The tumor is composed of many cysts of varying sizes and lined by endothelium. It may contain clear watery fluid or fluid showing evidence of hemorrhage or

Fig. 24. Cystic hygroma extending around the cervical viscera and into the floor of the mouth. Tracheotomy was necessary immediately after the infant was born. (Courtesy of Dr. S. Perzik.)

secondary infection. Cystic hygroma may attain great size (Fig. 24) and extend around the cervical viscera and into the floor of the mouth or distally into the mediastinum, occasionally producing severe respiratory distress by pressure on the larynx or trachea. Sometimes an asymptomatic hygroma may suddenly become obstructive, owing to hemorrhage or infection of the tumor.

The treatment of cystic hygroma is surgical. Aspiration, injection of sclerosing agents and radiation have all been used with generally undependable or unsatisfactory results. When unsuccessful, these make subsequent operation much more difficult. Certainly, in the face of obstructive symptoms, surgery is the only treatment recommended,

and sometimes preliminary tracheotomy is lifesaving (Fig. 24). Occasionally aspiration of cysts may produce temporary relief while preparing for operation.

At operation a thorough knowledge of the anatomy of the neck is extremely important, since most of the tumors are closely associated with the carotid vessels, jugular vein and many important nerves. As much of the tumor as possible should be removed with safety, and any remaining cysts should be unroofed.[11] These growths are not malignant, and the tendency to spread is minimal, so that the goal of surgery is to relieve or prevent respiratory obstruction and to achieve a good cosmetic result.

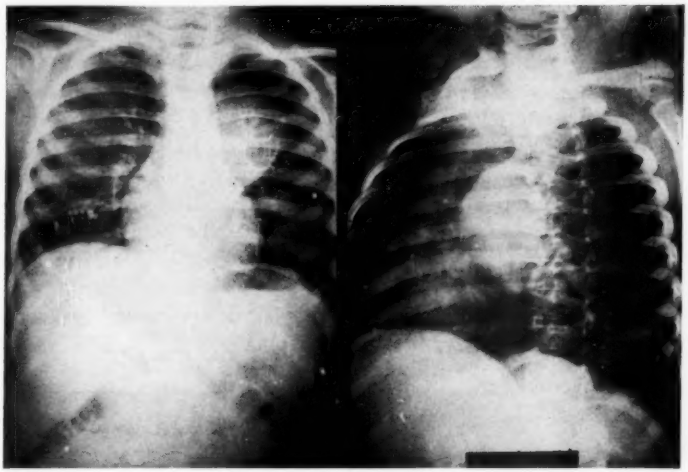

Fig. 25. Neurofibroma located in the posterior mediastinum, appearing as a rounded, sharply defined homogeneous shadow.

Congenital goiter with suffocation has been described by Handlesman and Sussman.[5] Partial thyroidectomy is recommended rather than irradiation or tracheotomy on the basis of previous reports in the literature. This condition seems to be increasing in frequency because of the administration of thiourea derivatives to pregnant women.

The diagnosis of *mediastinal cysts and tumors* is established by roentgenograms, and the various types of lesions can frequently be differentiated by their position in the chest. Neurogenic tumors are usually in the posterior mediastinum (Fig. 25). Goiters and thymomas are in the anterior superior mediastinum. The terato-dermoid group are found in the anterior inferior mediastinum and therefore rarely produce respiratory obstruction unless they reach a large size. Lymphomas,

bronchogenic cysts and esophageal duplications are most frequently located in the hilar area.

The most common of the benign *neurogenic tumors* is the neurofibroma, while the neuroblastoma predominates among the malignant tumors. Roentgenologically, these tumors appear rounded or elongated or lobulated and are sharply defined homogeneous shadows (Fig. 25). Calcification is sometimes present. There is occasional erosion of ribs or vertebrae, particularly by the malignant lesion. The prognosis is excellent if the lesion is benign. Neuroblastomas respond best to total removal and irradiation, although some survivals and apparent cures have been seen after incomplete surgical removal followed by irradiation.

Not all enlarged thymus glands are to be blamed for obstructive symptoms. A true thymoma, however, has produced symptoms which were relieved upon removal.

Teratomas (see also p. 1209) and *dermoids* are usually roentgenographically discrete, circular or globular tumors of homogeneous density. Calcium, plaques of bone, and teeth are occasionally seen.[8]

Bronchial cysts are characterized by a lining of ciliated pseudostratified columnar epithelium. They are closely associated with the respiratory passages, commonly lying near or above the bifurcation of the trachea. As they enlarge, obstructive symptoms appear and increase.[8]

Duplication of the esophagus is represented by a cyst adjacent to the esophagus and having a common muscular wall with the esophagus. The lining membrane may have gastric mucosa, and this may produce

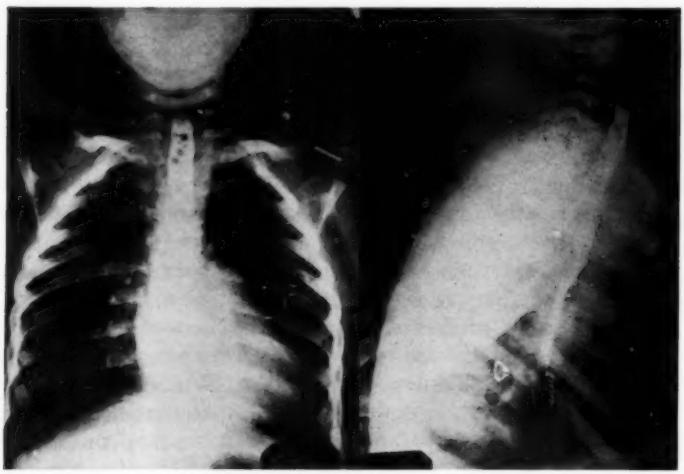

Fig. 26. Vascular ring. A swallow of Lipiodol shows indentation of the posterior and right lateral walls of the esophagus.

an acid fluid in the cyst. Complications of this lesion may be ulceration, hemorrhage, perforation into one of the surrounding structures, dysphagia and respiratory obstruction. Roentgenologically, one sees a large, smooth mediastinal mass, which on an esophagogram may deviate the esophagus from the midline. Treatment is surgical removal when discovered.

Vascular rings (see also p. 1057) *or anomalies of the aortic arch* produce respiratory distress by external pressure on the trachea. Symptoms vary from an inspiratory stridor and an odd cough to suffocation. A swallow of Lipiodol will show indentation of the posterior wall of the esophagus (Fig. 26). It is sometimes possible to demonstrate narrowing of the trachea on a good plain roentgenogram, and endoscopic examination may show a vessel pulsating against the posterior wall of the esophagus. Treatment is early division of the vessel producing the obstruction. Too long a delay will sometimes produce permanent impairment of the trachea at the pressure point.

ESOPHAGEAL ATRESIA

Esophageal atresia (see also p. 958), with or without the various types of tracheo-esophageal fistula, is one of the most common lesions producing respiratory distress which require surgery. About 90 per cent of the cases are represented by a blind-ending upper esophageal segment and a connection between the trachea and lower esophageal segment. Another 5 per cent is represented by the "pure" esophageal atresia not associated with tracheal connections. The remaining 5 per cent comprise the rarer forms of tracheo-esophageal fistula without esophageal atresia (H-type), fistula between upper esophageal segment and trachea, and connections between the trachea and both upper and lower segments (Fig. 27).

Although delay in diagnosis was the rule in the past, increasing knowledge of this condition has made common earlier discovery and treatment. Indeed, with the increasing use of the highly-to-be-recommended delivery room procedure of immediate aspiration of gastric contents after delivery, many cases are being demonstrated in the first few minutes of life.

The most common symptom is that of a "wet mouth" in the newborn because he is unable to swallow his own saliva. This is accentuated when the first attempt at oral feeding is made. The infant will choke and cough, and cyanosis occurs, relieved only by suction or more coughing. The diagnosis is established by inability to pass a catheter into the stomach and by roentgenograms taken after injection of radiopaque material through the catheter. A no. 10 or 12 French soft rubber catheter passed through the mouth is safer than the newer

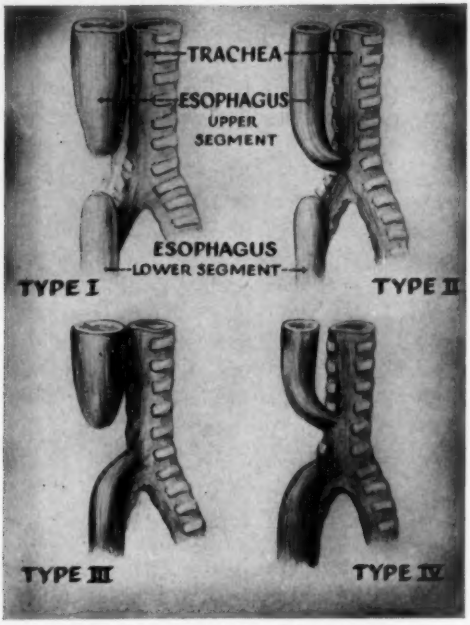

Fig. 27. Atresia of the esophagus. Type I, without fistula, comprises about 5 per cent of all cases (see Fig. 28). Type III, about 90 per cent of all cases, has a blind-ending upper segment of esophagus and a lower segment communicating with the trachea (see Fig. 29). All other types amount to the remaining 5 per cent. H-type of fistula without atresia is not illustrated above.

stiffer plastic catheters. Auscultation of the upper part of the chest and abdomen while a few cc. of air are injected into the catheter will assist in determining whether it has passed into the stomach. On the anterior-posterior chest film one can often see the air-filled, dilated upper esophageal segment standing out in contrast to the surrounding organs. However, the catheter should be aspirated carefully and 1 cc. of contrast material injected into the upper esophagus. It will demonstrate an upper blind pouch or an upper fistula with the trachea. The presence or absence of air in the intestines will demonstrate the presence or absence of a connection between the trachea and distal esophageal segment (Figs. 28, 29). The abdomen is scaphoid in the absence of intestinal air. The presence of atelectasis and/or pneumonia should be determined by the same roentgenograms.

When the tracheo-esophageal opening is large, the infant may have a sustained cyanosis secondary to abdominal distention. This is not uncommon and occurs when, with each breath the infant expires, especially when crying against partially closed vocal cords, almost as much air is forced through the fistula as outward through the pharynx.

These infants will have a weak cry and will often pass flatus within an hour or so of birth.

Careful examinations should be carried out, looking for associated anomalies, the most common being anorectal, genitourinary and car-

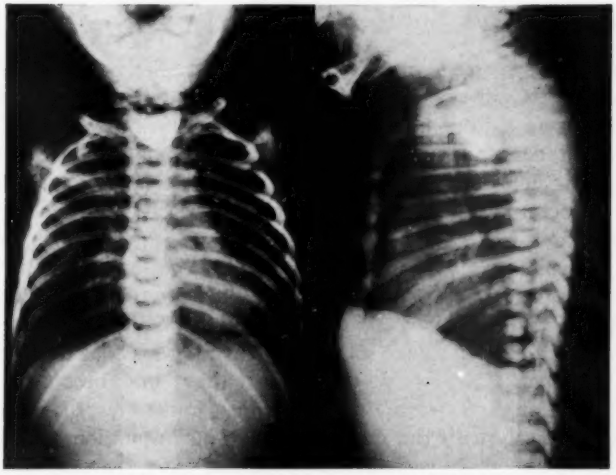

Fig. 28. Atresia of the esophagus without tracheo-esophageal fistula. Lipiodol in upper blind-ending segment. Absence of gastrointestinal air indicates absence of fistula.

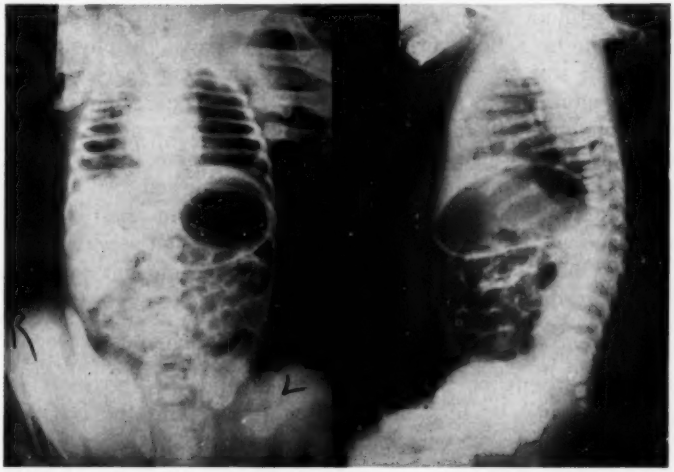

Fig. 29. Atresia of the esophagus with tracheo-esophageal fistula. Lipiodol demonstrates upper blind-ending segment. Presence of gastrointestinal air indicates a communication between the trachea and the lower esophageal segment.

diac. Preoperatively the pharynx should be kept clear by frequent aspiration. Any dehydration or pneumonia should be treated with appropriate fluids or antibiotics. Surgical repair should be carried out as soon as the infant is stabilized.

PULMONARY CYSTS

Pulmonary cysts (see also p. 1001) may be congenital or acquired, single or multiple. Cysts occurring in newborn infants, or in older infants not having an episode of previous pulmonary infection, may usually be considered congenital cysts. Circumscribed radiolucent areas appearing in lung fields after respiratory infections, and most frequently after staphylococcal pneumonia, are considered to be postinfectious pneumatoceles[12] or acquired cysts (see also p. 1046). Congenital cysts are lined with a respiratory type of epithelium and are filled with either fluid or air. If a communication is present with the respiratory tract, fluid-containing cysts usually evacuate and become filled with air. If this communication is freely patent, the lesion will behave as a nonexpanding cyst. Such cysts may disappear in time, or may become involved with infection and become complicated. Sometimes the cyst communicates freely with the respiratory tract on inspiration, but exhibits a valvelike action. In such instances air can enter the cyst easily, but cannot escape, and a dramatic sequence of events occurs. As the cyst enlarges, it compresses or collapses the rest of the lobe and the lung which contains it. Then progressively the diaphragm is depressed and the mediastinum is pushed to the opposite side with compression of the opposite lung. Such an infant is in dire straits. On physical examination one sees an infant with dyspnea of varying degrees, but as it becomes more severe it is followed by cyanosis and tachycardia. On the affected side there is hyperresonance to percussion, and breath sounds are diminished or absent. The mediastinum is shifted to the opposite side, where breath sounds are diminished. An immediate anteroposterior and lateral roentgenogram of the chest should be made. One will see a large cystic cavity, compression atelectasis of the remaining lobe or lobes, and depression of the diaphragm on the same side. The mediastinum will be displaced toward the opposite side with compression of that lung (Fig. 30).

It is imperative to remove the space-occupying lesion so that the collapsed portions of the lung can re-expand. In the most severe instances it may be lifesaving to aspirate air from the cyst with a needle and syringe, or with a needle and water-seal apparatus, while making urgent preparations for surgery. When this is done, the physician must stay with this patient constantly until operation is performed. Oxygen should also be given.

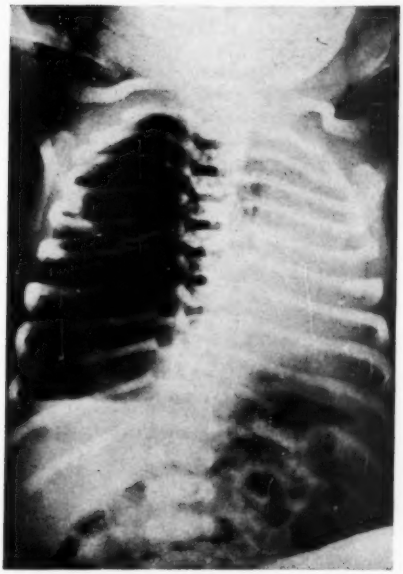

Fig. 30. Preoperative roentgenogram of the chest of a 5-day-old infant with a congenital lung cyst. In addition to the large cystic cavity, one sees depression of the right diaphragm, shift of the mediastinum to the left side, and compression atelectasis of the remaining lobes.

Because of the reduced amount of functioning lung, pulmonary exchange will be minimal, and the anesthesiologist may have difficulty in producing effective anesthesia. If air is forced into the lungs by vigorous assistance, more air may be forced into the cyst, further compromising the vital capacity. On such occasions it may be wise to open the chest with local anesthesia. Once the chest is open, the cyst or lobe will push itself out or can be easily delivered. Now inhalation anesthesia can be accomplished without difficulty and the lungs expanded. The cystic area can be removed by segmental resection or by lobectomy in most instances. As much normal lung as possible should be saved. Water-seal drainage, oxygen and humidity are used postoperatively.

When asymptomatic congenital lung cysts are not treated, there is considerable danger should the cyst become infected or expansile.

On the other hand, surgical removal of an uncomplicated cyst in a healthy infant is a relatively simple procedure. For these reasons we recommend that all *congenital* lung cysts should be removed when discovered. Postinfectious pneumatoceles, on the other hand, usually disappear spontaneously. The treatment of the recurrent or complicated pneumatocele is not within the scope of this paper.

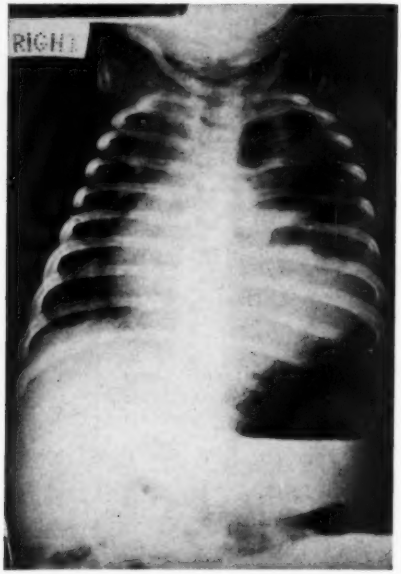

Fig. 31. Chest roentgenogram of a 5-month-old infant with respiratory distress, fever and failure to gain weight. The upper half of the left lung appears cystic, and the lower half is partially atelectatic. Subsequently an anomalous lobe of lung arising from the esophagus was removed, resulting in a cure.

One of us[3] described an anomalous lobe of lung arising from the esophagus and producing respiratory distress (Fig. 31). Removal of the lobe resulted in a cure.

OBSTRUCTIVE PULMONARY EMPHYSEMA

This is a special type of pulmonary emphysema confined to one segment, lobe or lung, and is due to a valvular mechanism by which air can enter this part freely, but cannot exit. This has been reported due to valvelike folds of mucosa in the lobar bronchus, deficiency of the cartilaginous rings of the bronchus, a persistent ductus arteriosus, and herniation of lung through an anterior mediastinal defect.[2, 7] Some proved cases have no proved etiology.

As the segment or lobe becomes more and more blown up, it crowds the remaining lung tissue, and there is less room for pulmonary air exchange. The previously well infant or young child will suffer dyspnea and later cyanosis and tachycardia. Wheezing may or may not be present. The similarity between this condition and expanding lung cyst is evident. Physical examination reveals hyperresonance and di-

minished breath sounds over the affected area and shift of the mediastinal structures toward the opposite side. Anteroposterior and lateral roentgenograms show a large area of radiolucency which can usually be differentiated from pneumothorax or lung cyst by the demonstration of faint lung markings throughout the radiolucent area. In addition, similar to the lung cyst, the remaining lung on the affected side is compressed or atelectatic, the diaphragm is depressed, and the mediastinum is shifted toward the opposite side, where the lung may be compressed (Fig. 32). Bronchoscopy may be of value in excluding

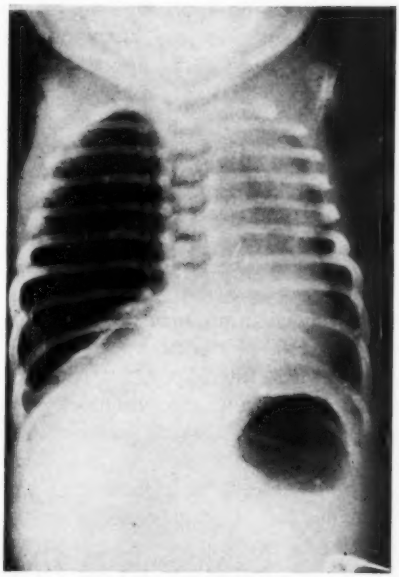

Fig. 32. Preoperative chest roentgenogram of a 17-day-old infant with emphysema of the right upper lobe. Faint lung markings can be seen throughout the emphysematous lobe. The right diaphragm is depressed, the mediastinum is shifted to the left, and the collapsed middle and lower lobes are seen against the diaphragm.

or removing intraluminal causes of emphysema, the most important and most frequent of which is foreign body. In the most critical cases, removal of air by needle and syringe or water-seal apparatus may be temporarily lifesaving while urgent preparations for surgery are being made and the child is being taken to the operating room.

The anesthetist may be faced with the same problem of insufficient pulmonary exchange as he faces with the expanding lung cyst, and the answer is the same: If anesthesia cannot be accomplished in the usual way, open the chest under local anesthesia, and the emphysematous

lobe will push itself out or be easily delivered. Then the lungs can be expanded and anesthesia accomplished. Lobectomy is the usual procedure and is curative.

PNEUMOTHORAX AND PNEUMOMEDIASTINUM

Increased intra-alveolar pressure from any cause may produce alveolar rupture and pneumothorax. Air dissects along the perivascular sheaths and enters the lung roots and mediastinum and may rupture into the pleural cavities. Positive pressure resuscitative measures are often incriminated, and vigorous crying and coughing are also suspect.

Mild degrees of pneumothorax and pneumomediastinum are probably more common than is generally appreciated and may cause no respiratory embarrassment. Lack of symptoms results in failure to demonstrate the frequency of this lesion. Large accumulations of air in the pleural space may cause dyspnea, cyanosis and tachycardia. Physical examination reveals hyperresonance and diminished breath sounds on the affected side with limitation of movement of the chest. The heart and mediastinum can be demonstrated to be displaced toward the opposite side, and the diaphragm (and liver, if on the right side) is displaced downward.

Extensive pneumomediastinum may cause bulging of the sternum and distention of the veins of the neck. A large enough collection of air behind the sternum may interfere with return of blood to the heart by the mechanism referred to as the "air block syndrome."

The diagnosis is established by anteroposterior and lateral roentgenograms of the chest. In pneumothorax one sees a radiolucent band of varying width around the lung. If the amount of air is large and under tension, the underlying lung is compressed, the diaphragm is pushed downward, the mediastinum is displaced toward the other side, and the other lung may thus be also compressed (Fig. 33). If fluid is also present, an air-fluid level may be seen in the upright view. In pneumomediastinum one sees a longitudinal radiolucent band along the borders of the mediastinum in the frontal view and a localized collection of air in the anterior mediastinum in the lateral view. Ledner[6] has described the "flying thymus" sign in which the lobes of the thymus are displaced upward, laterally, and usually anteriorly, resembling wings in flight (Fig. 34).

If the pneumothorax or pneumomediastinum is small and the symptoms are minimal, treatment is limited to close and careful observation. Larger collections of air should be aspirated with a syringe and needle followed by an immediate check roentgenogram and careful observation. If this procedure produces no improvement after one repetition, or if there is any evidence of tension, an intercostal tube

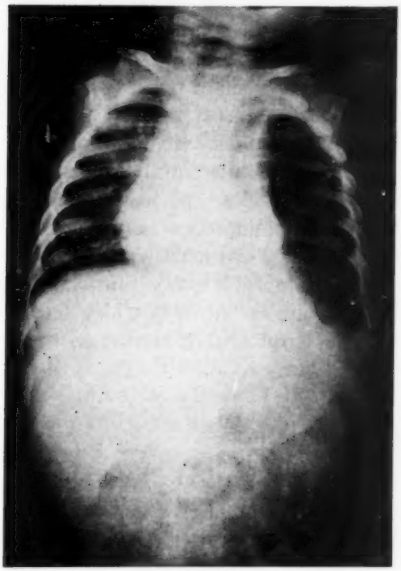

Fig. 33. Chest roentgenogram of a 10-month-old infant with a left pneumothorax following staphylococcal pneumonia, showing an air-filled pleural cavity with the lung collapsed about the hilus and diaphragm. The left diaphragm is depressed, and the mediastinum is displaced to the right.

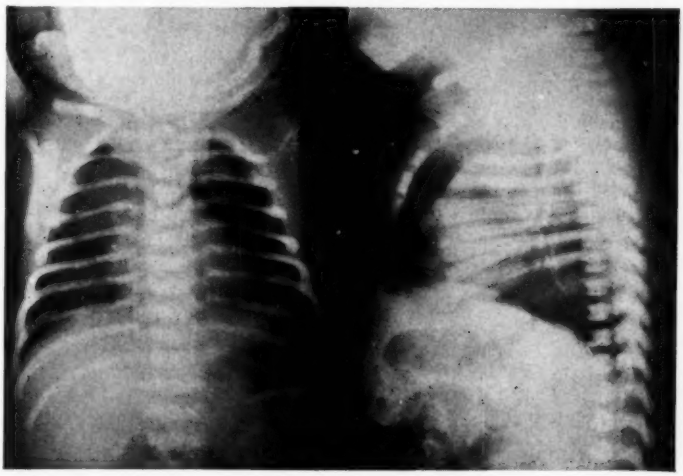

Fig. 34. Chest roentgenograms of a newborn infant with pneumomediastinum. Radiolucent bands are seen on each side of the mediastinum, and a localized collection of air is demonstrated behind the sternum. The lobes of the thymus are displaced upward, the "flying thymus" sign described by Ledner.

should be inserted laterally through the fifth or sixth intercostal space and connected with a water-seal apparatus. For this purpose it is best to use a no. 12 or 14 French catheter inserted through a trocar. This procedure should produce immediate improvement, and the air should stop bubbling through the water-seal within 12 to 24 hours, indicating that the leak is closed. If the leak remains open after this period, suction should be used on the intercostal tube, and thoracotomy may become necessary to find and close the leak under rare circumstances.

A frequent cause of pneumothorax in infancy is associated with pulmonary infectious disease, particularly staphylococcal pneumonia. The treatment of this condition is not within the scope of this paper (see p. 1043) except in those instances in which tension pneumothorax exists. On such occasions treatment is similar to that for spontaneous pneumothorax (Fig. 33).

CONGENITAL DIAPHRAGMATIC HERNIA

Hernia through the diaphragm occurs in three general areas. Most commonly involved is the posterolateral area known as the foramen of Bochdalek. Less commonly involved is the substernocostal area (foramen of Morgagni), and the esophageal hiatus. Respiratory distress is produced when a sufficient volume of intra-abdominal organs is present in the chest to encroach upon respiratory space. This rarely

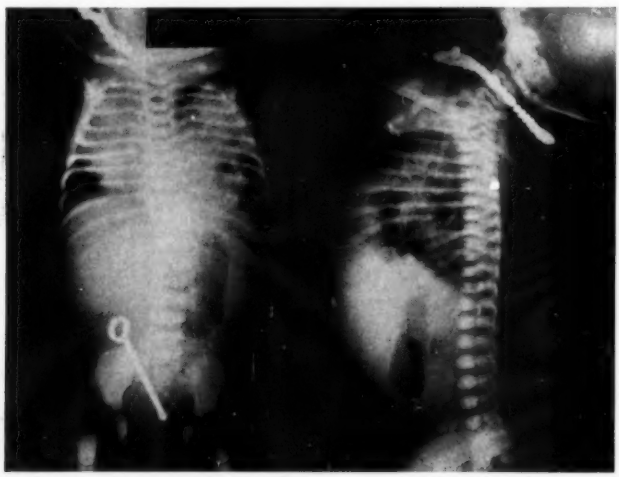

Fig. 35. Chest roentgenograms of a newborn infant with a diaphragmatic hernia through the foramen of Bochdalek. Air-filled loops are seen in the left chest, and the abdominal cavity is relatively empty. The spleen is near the apex of the pleural cavity. The mediastinum is severely shifted to the right.

occurs in the latter two types of hernia, but is common in the Bochdalek variety, particularly when the stomach and intestines fill with air. For this reason it is sometimes lifesaving to pass a nasal-gastric tube for the relief or prevention of gaseous distention, as soon as the diagnosis is made, while preparations are made for immediate operation.

Physical examination reveals cyanosis and rapid respirations, scaphoid abdomen, and characteristic findings in the chest. Percussion and auscultation reveal a shift of the heart and mediastinum to the opposite side and diminished breath sounds on the affected side. There may be either dullness or tympany over the herniated organs, depending on whether or not they are distended with air. Auscultation of peristaltic sounds in the chest is not a common or reliable sign in the newborn. Roentgenograms without contrast media will confirm the diagnosis. Gas-filled loops will be seen in the chest, frequently with communication through the diaphragm. The lung on this side will be collapsed, and the mediastinum will be seen shifted to the opposite side (Fig. 35). (See also p. 946.)

Immediate operation is indicated to prevent or to relieve symptoms. High mortality rates occur when operation is delayed. We believe that the approach in newborns should be through the abdomen because (1) reduction of the hernia is easier, (2) repair is easier because the organs can be eviscerated to facilitate exposure, (3) other intra-abdominal anomalies, which are common, can be recognized and treated, and (4) lack of adequate intra-abdominal space for the replaced organs can be accommodated for by closing only skin and subcutaneous tissues and thereby establishing an abdominal wall hernia. The thoracic route may be advantageous in older infants or children and occasionally in the less common right-sided hernias when the liver may block exposure of the diaphragm from below.

After reduction of the hernia the lung can expand and the mediastinum return to its normal position. Not infrequently the anesthetist will not be able to expand the lung with moderate pressure. More vigorous pressure may produce a rupture in this or the contralateral lung. It is better to allow the hypoplastic lung to expand spontaneously in the next few days or weeks.

Eventration of the diaphragm implies an abnormally high but intact diaphragm. This lesion may be congenital in that there is a lack of muscle between the pleural and peritoneal reflexions; or it may be acquired and caused by a birth injury involving the phrenic nerve which paralyzes one leaf of the diaphragm.[1] As the intestinal tract becomes normally distended with gas, the diaphragm rises and encroaches upon the breathing space. In addition, the mediastinum shifts to the opposite side, encroaching upon its volume for expansion, and compression

atelectasis occurs. In instances of more than mild severity the infant will exhibit tachypnea, dyspnea and cyanosis.

Roentgenographic studies will show elevation of the affected diaphragm, mediastinal shift, pulmonary compression, and paradoxic motion of the involved diaphragm (Fig. 36).

Treatment consists in surgical plication of the diaphragm by a thoracic approach. We have seen one infant die when operation was delayed.

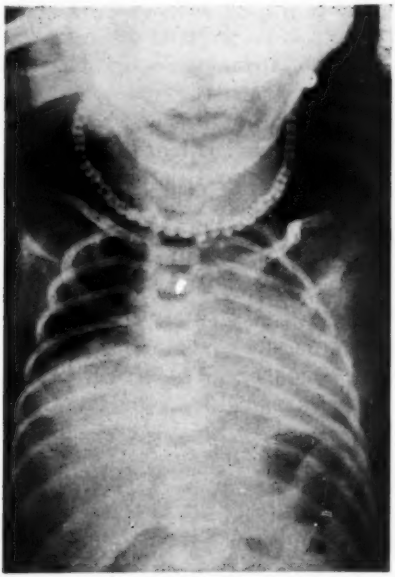

Fig. 36. Chest roentgenogram of a newborn infant with congenital eventration of the diaphragm. In addition to considerable elevation of the entire right diaphragm, there is pulmonary compression and mediastinal shift.

SUMMARY

Respiratory obstructions in infants and children are frequently urgent situations requiring the utmost in care and skill and leaving little time for delay in recognition and treatment.

The diagnosis is quickly established by evaluation of the cry and respirations, examination of the head, neck and chest, and interpretation of properly taken roentgenograms.

Various causes of respiratory obstruction of surgical import are discussed, with emphasis on recognition and general principles of treatment.

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STAPHYLOCOCCAL PNEUMONIA

Pleural and Pulmonary Complications

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Today many of the deaths which occur in infants and the occasional child afflicted with acute necrotizing staphylococcal pneumonia appear to be due to failure to deal successfully with the surgical complications of this disease rather than to the pulmonary infection per se. Pyopneumothorax, empyema, tension pneumothorax and, occasionally, pneumatocele or intrapulmonary abscess are surgical emergencies which should be anticipated by the responsible physician so that they may be managed early and adequately. Fibrothorax and small, frequently multiple pneumatoceles, by contrast, rarely require surgical treatment, since they may be expected to resolve spontaneously within a few months after the intrapulmonary infection has been controlled.[2] Since staphylococcal pneumonia carries a high mortality and morbidity, it is believed that further improvements in therapy may best be served by a review of the problem as presented by a series of cases admitted to the Columbus Children's Hospital during the past five years. In this period there have been 39 cases of bacteriologically proved staphylococcal pneumonia.

Twenty-five of these were considered to be *primary*. The initial complaint was referable to the pulmonary infection, although in several instances impetigenous lesions of the skin had previously been noted. The remaining 14 cases are grouped together as *secondary* staphylococcal pneumonia, since in each instance the patient was known to

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have a separate focus of active infection, for example, an unhealed burn, osteomyelitis, soft tissue abscess or myelomeningocele prior to the development of pulmonary infection and subsequent complications.

Eighteen of the 39 patients died, a mortality rate of 46 per cent. Autopsy was performed on all patients who died. Six of these patients were found to have surgically remediable complications which were clinically unrecognized or inadequately treated. By contrast, there were no deaths in 13 cases in which surgical complications were suspected sufficiently early so that adequate treatment could be carried out.

The bacteriologic studies appeared to be of prognostic significance in this entire group, particularly as far as surgical complications are concerned, since, of the 22 patients who were phage-typed, 16 had infections due to the 80–81 phage strain of Staphylococcus. All these patients had either empyema or pyopneumothorax during the course of their illness, and many also subsequently were demonstrated to have pneumatoceles.

PRIMARY STAPHYLOCOCCAL PNEUMONIA

Age Incidence

Twenty-four of the 25 patients with primary staphylococcal pneumonia in this series were less than 2 years of age; 19 were less than 6 months; and 8 were under 1 month at the time of their admission (Fig. 37).

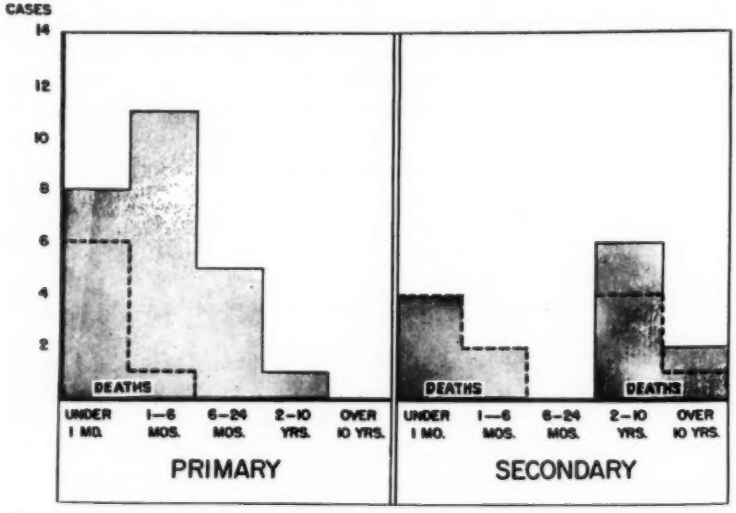

Fig. 37. Age incidence and mortality of 39 cases of primary and secondary staphylococcal pneumonia.

Epidemiology

In 10 patients it appears likely that the infection was acquired as a result of a previous hospitalization, either in a newborn nursery where virulent staphylococcal strains had recently been isolated from patients and personnel, or from the infant ward areas of the Columbus Children's Hospital. Five subjects had been previously hospitalized in this hospital for noninfectious conditions within two weeks prior to the onset of their pneumonia.

Signs and Symptoms

Many of these infants and children had symptoms of upper respiratory tract infection, including coryza, cough and low-grade fever of intermittent type for 4 to 14 days prior to the time of their hospitalization with pneumonia. Usually, one or two days before admission, however, there was a noticeable increase in respiratory difficulty, including rapid labored respirations and occasional cyanosis, at which time the fever usually rose and became constant.

By the time of admission more than half of the patients were described as having grunting, rapid, shallow respirations indicative of acute ventilatory distress and suggestive of pleuritic pain. Eight were visibly cyanotic. Four were in shock, all of whom eventually died. The admission temperature varied between 101° and 103° F. in all except 5 patients who had normal or subnormal temperatures.

Laboratory Studies

Chest films at the time of admission demonstrated unilateral pulmonary infiltration in most cases. The diagnosis of staphylococcal pneumonia was suspected by the radiologist early in the course of hospitalization in 10 cases when repeat chest films showed evidence of pneumatocele or pyopneumothorax.

Bacteriophage typing was performed on 17 patients, in 14 of whom the infection was due to the 80–81 strain. Two cases were due to phage type 83 (formerly typed as VA4, 75, 44A), and in the remaining case the organism was not specifically typable.

Surgical Complications

The commonest surgical complication encountered was *empyema* (Fig. 38). This was present at the time of admission or developed during the hospital stay in 17 patients (68 per cent). In only one case was it bilateral. Eight of these patients, or 32 per cent of the entire group, also had demonstrable free air in the pleural space associated with the empyema (pyopneumothorax).

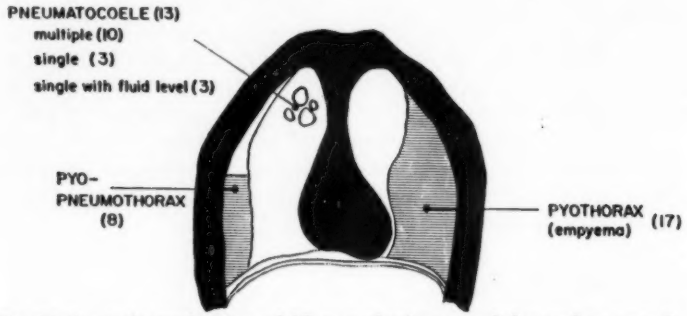

Fig. 38. Surgical complications of 25 cases of primary staphylococcal pneumonia.

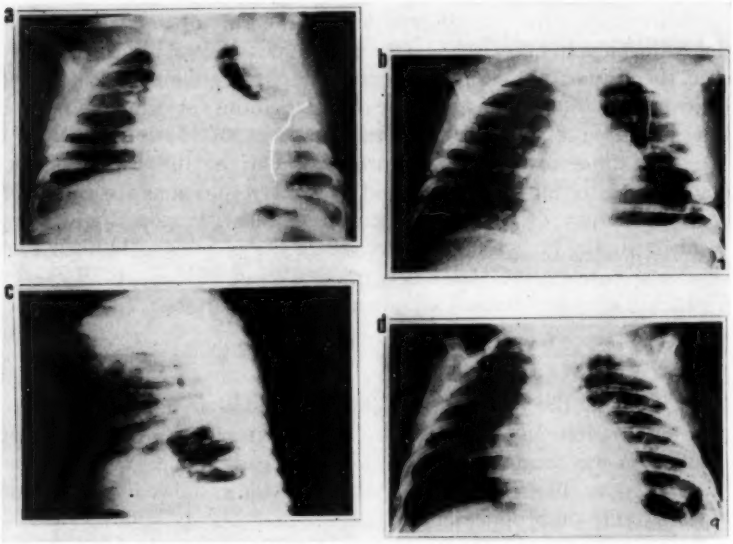

Fig. 39. Infected pneumatocele in a 3-month-old, white female infant, treated by open thoracotomy drainage. *A*, (3/16/59) Pyopneumothorax with small pneumatocele, left base. Discharged from outside hospital 2 days later. Re-admitted 3/28/59 with spiking fever. *B*, *C*, (3/31/59) Admission films, Columbus Children's Hospital. Large pus-filled pneumatocele. *D*, (4/6/59) Four days after open thoracotomy (Monaldi) drainage. Lung completely expanded. Temperature normal. Pus from pneumatocele cultured *Staphylococcus aureus*, phage type 80–81.

Pneumatoceles, the second most common surgical complication, tended to develop somewhat later in the course of the disease and could be demonstrated on serial radiographic studies in 13 (52 per cent) of the patients. In 10 instances the air cysts were multiple, and in only one case were they bilateral. In 4, fluid levels in the pneumatoceles were noted, and in 2 of these a thoracotomy for drainage was

performed (Fig. 39). Eleven of the patients with pneumatocele had empyema, and two had pyopneumothorax. In a single patient rupture of a pneumatocele led to the subsequent development of a tension pneumothorax while the child was at home one month after the original demonstration of a pneumatocele on the chest film. This child survived and recovered after closed intrapleural thoracotomy (Fig. 40).

In summary, surgical complications developed in 19 of these 25 patients (76 per cent) with primary staphylococcal pneumonia.

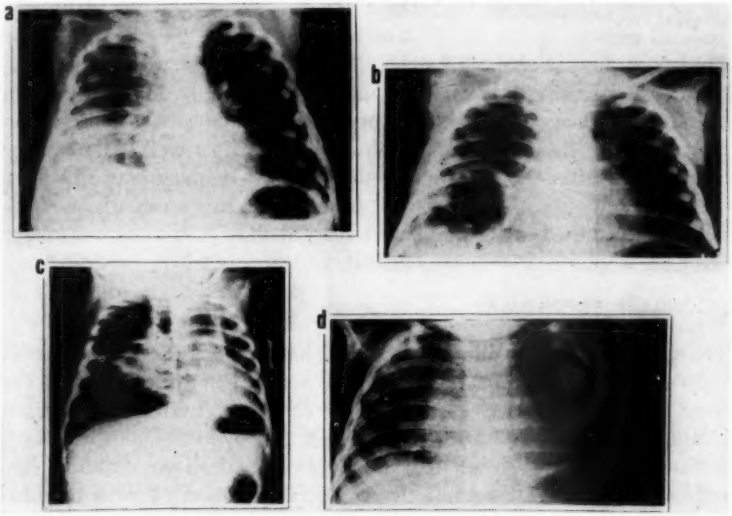

Fig. 40. Tension pneumothorax in a 4-month-old white female infant, following rupture of a pneumatocele. *A*, (12/20/56) Admission film. Right upper lobe and right lower lobe, pneumonia; empyema. *B*, (1/6/57) Pneumonia clearing. Two pneumatoceles present on the right. Patient asymptomatic. *C*, (2/6/57) Tension pneumothorax with mediastinal shift, occurred at home. Closed tube thoracotomy drainage immediately performed upon readmission. *D*, (4/2/57) Normal chest film. Six weeks after drainage discontinued.

Mortality

Seven (28 per cent) of the 25 children died (Fig. 37). Six of the deaths were in infants less than one month of age, and the seventh occurred in a seven-week-old infant.

Medical Treatment

Medical treatment consisted in supporting the circulating blood volume, providing a humidified oxygen-rich atmosphere and the adminis-

tration of a wide variety of antimicrobial agents. Thirteen patients had medical management alone; seven of these died. Of particular interest is the fact that five of the infants who died had an inadequately treated empyema, and these deaths must therefore be regarded as possibly preventable. In one instance delayed recognition of an acute tension pneumothorax in a four-week-old infant with a known but undrained empyema was responsible for her demise. The average hospital stay of the 6 surviving patients was 26.8 days.

Surgical Treatment

Twelve of the patients with surgical complications of pneumonia were treated shortly after the diagnosis had been made by closed thoracotomy tube drainage (10 cases) or open thoracotomy with rib resection (2 cases). All these children survived and are now well. The average hospital stay after the initiation of thoracotomy drainage was 18.3 days.

Fortunately, fibrothorax was not observed in this series of cases following either medical or surgical management.

SECONDARY PNEUMONIA

There were 14 infants and children who had staphylococcal pneumonia secondary to a recognized, pre-existing source. In four instances the focus was a soft tissue abscess in an otherwise healthy child. Four infants had an infected myelomeningocele and hydrocephalus for which a ventriculojugular shunt had been performed. Two children had unhealed burns, two had soft tissue abscesses associated with terminal malignancy, and two suffered from osteomyelitis.

Age Incidence

There was a wide range of age distribution in the secondary group in contrast to the patients with primary staphylococcal pneumonia (Fig. 37). Four of the patients were less than one month of age, and eight were older than two years.

Epidemiology

In nine cases the staphylococcal infection was considered to be hospital-acquired. All these patients died, and in no case was the diagnosis of staphylococcal pneumonia made before death. None of these patients had surgical complications; in 4 the phage type was 83, and in 5 it was not known.

Surgical Complications

Surgical complications occurred in four of the patients with secondary infections, all of whom had *empyema* (two with *pyopneumothorax*). *Pneumatoceles* coexisted with empyema in two patients. In 2 of the patients the phage type was 80–81, in 1 it was not typable, and in 1, unknown.

Mortality

Eleven of the 14 patients (78 per cent) died. The three survivors all had empyema, and closed thoracotomy drainage was performed on one. One apparently preventable death occurred in a child who died with an undrained empyema following the development of an acute tension pneumothorax.

Surgical Treatment

It would appear that if the mortality and morbidity of staphylococcal pneumonia are to be improved, the simple surgical procedures required for this disease should become part of the armamentarium of all physicians. It seems worth while, therefore, to describe in some detail the equipment and technique which may be used to treat empyema, pneumothorax, tension cysts or intrapulmonary abscess.

The majority of such procedures can be performed safely in a well equipped and attended treatment room, usually using local anesthesia. The operator and his assistants should be appropriately gowned and gloved primarily for their own protection (Fig. 41).

THORACENTESIS. After preparation of the skin with a tincture solution such as Zephiran the subcutaneous tissue is anesthetized with an intradermal needle, after which procaine or Xylocaine is injected with a small intradermal needle. After this a short-bevel no. 22 needle is passed down to the rib and redirected above the rib into the intercostal space and beneath the endothoracic fascia for the introduction of more local anesthetic solution.

After a brief period to permit the anesthesia to take effect, a short-bevel no. 18 needle attached to a 3-way stopcock and 20-ml. syringe is passed through the previously anesthetized tract just above the rib to avoid damage to the neurovascular bundle until the endothoracic fascia and pleura are perforated. At this point, when pleural fluid or air is obtained, a hemostat is applied flush with the skin to prevent any further penetration of the needle while subsequent aspiration is carried out (Fig. 42).

Any material aspirated from the pleural cavity is placed immediately in a sterile tube for smear and culture studies. If no fluid is obtained, the needle is placed in the culture tube for similar purposes.

CLOSED THORACOTOMY. If closed thoracotomy drainage is to be performed, a small skin incision 3 to 4 mm. in length is made and an appropriately sized sheathed trocar inserted into the pleural cavity. The trocar is removed, and a stiff, multiholed urethral catheter which has been appropriately marked so that it will not be inserted more than 1 or 1½ inches into the intrapleural space is then passed through the sheath into the chest and the sheath removed. The catheter may be maintained in place with a

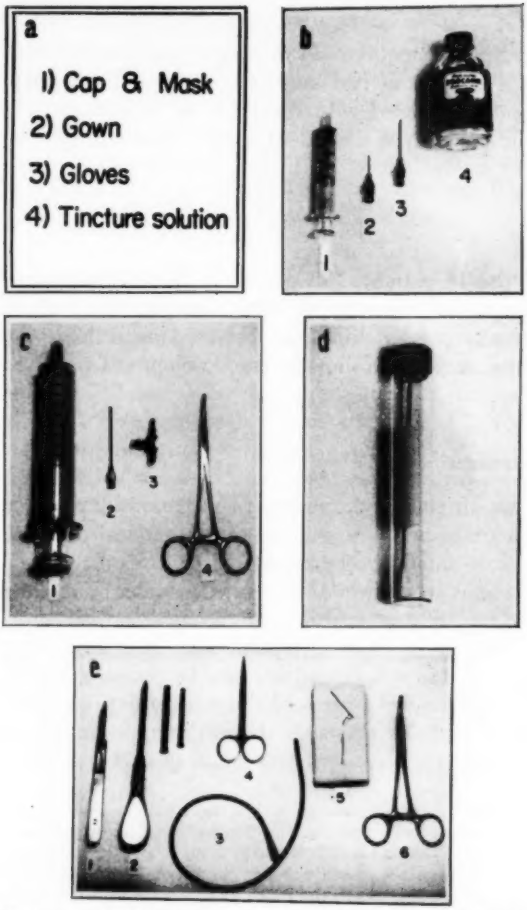

Fig. 41. Equipment for thoracentesis and closed thoracotomy drainage. *a*, Preparation of the operator and the skin. *b*, Local anesthesia: (1) 2-ml. syringe, (2) no. 25 needle, (3) no. 22 short-bevel needle, (4) procaine or Xylocaine, 1 per cent. *c*, Thoracentesis set: (1) 20-ml. syringe, (2) no. 18 short-bevel needle, (3) 3-way stopcock, (4) hemostat. *d*, Culture tube. *e*, Closed thoracotomy drainage set: (1) scalpel, (2) trocar (sizes 10 F, 14 F, 16 F), (3) multilumen catheters (sizes 10–12 F, infants; 14–16 F, children), (4) suture scissors, (5) cutting edge skin needle with 4-0 silk, (6) hemostat.

hemostat until it is sutured with a single stitch anchoring it to the skin. The catheter is then connected to the previously assembled 3-bottle drainage system, usually with a negative pressure of 12 to 15 cm. of water, which can be readily achieved with a Stedman type of pump (Fig. 43). Rarely will the bronchial-pleural leak be so large that the lung cannot be expanded with such a device, but if expansion is not obtained, oral-pharyngeal or wall suction will be required.

The chest tube should then be fixed to the chest wall with nonrestrictive adhesive

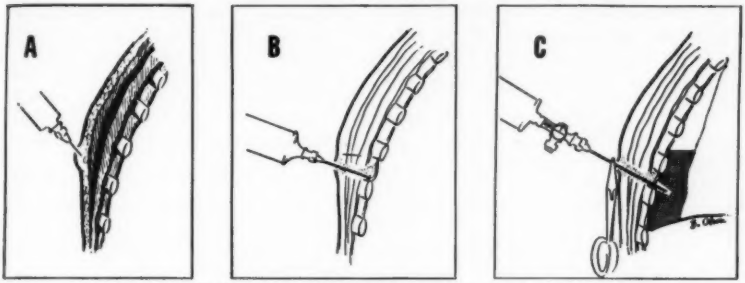

Fig. 42. Technique for thoracentesis. (See text for details.)

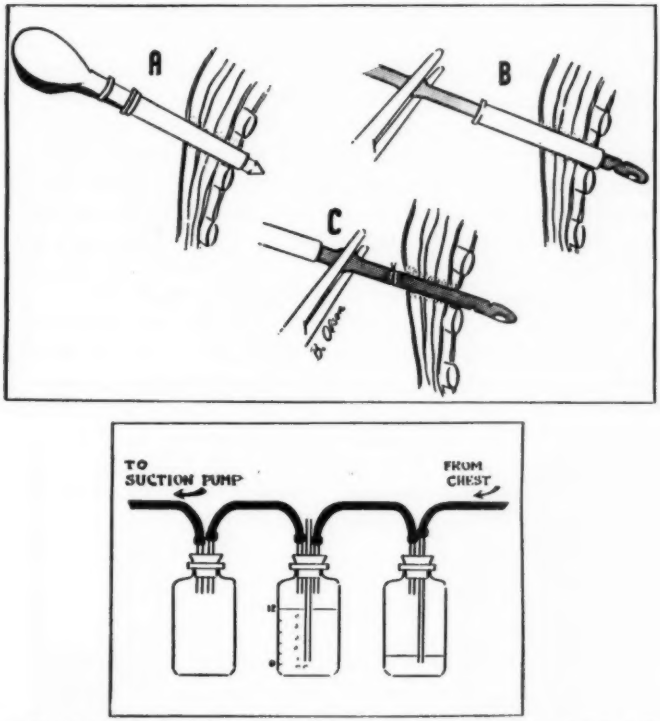

Fig. 43. Technique for closed thoracotomy drainage. (See text for details.) Bottle system equipment consists of 3 sterile, rubber-stoppered wide-mouth bottles, 7 pieces of glass tubing, 5 pieces of stiff connecting rubber tubing, 1 Stedman pump.

strapping adequately supported with sterile gauze to avoid kinking at the chest wall should the child change position.

Once the patient is connected to the drainage bottles, the bottles should be kept at least 2 feet below the chest level at all times. Such chest suction devices become an integral part of the patient and should not be clamped for routine nursing procedures or during transport of the patient to x-ray, operating room, and elsewhere.

Open Thoracotomy. Open thoracotomy drainage is a formal operative procedure that is rarely necessary except in the presence of loculated empyema. It is performed in a conventional manner by resection of a small portion of the fifth, sixth or seventh rib posteriorly, after which a large 18 to 26 French rubber catheter is inserted into the chest cavity after a surgical toilet of the pleural space to remove as much retained pus and fibrin as possible. The wound is tightly closed around the catheter, and an air seal is obtained by using a finger of rubber glove which is placed over the catheter and fixed to the skin with dermatome glue.

Drainage of lung abscesses is similarly performed. The chest is entered directly over the point where the abscess is closest to the thoracic wall. If the lung is firmly adherent to the chest wall, this procedure can safely be performed in one stage.

A Monaldi type of intracavitary drainage of large, tension lung cysts which, because of size or, rarely, chronicity, require decompression is in our opinion a more benign procedure than enucleation or pulmonary resection.

DISCUSSION

Experience at this hospital confirms the observations of others as to the high incidence of surgical complications of staphylococcal pneumonia.[1, 3, 4, 7] Empyema occurred in 68 per cent of the cases of primary pneumonia in this series and 71 per cent in the Boston Children's Hospital series of 79 cases. Such surgical complications can indeed be anticipated in nearly all cases in which a phage type 80–81 Staphylococcus is believed to be the pathogenic agent (Fig. 44). By contrast, surgical complications occurred in only 1 of 6 patients in whom a phage type 83 Staphylococcus was recovered. Eight of 17 patients in whom phage typing was not performed had empyema or pneumatoceles. There seems to be little doubt, therefore, that the development of such serious sequelae to necrotizing staphylococcal pneumonia as empyema,

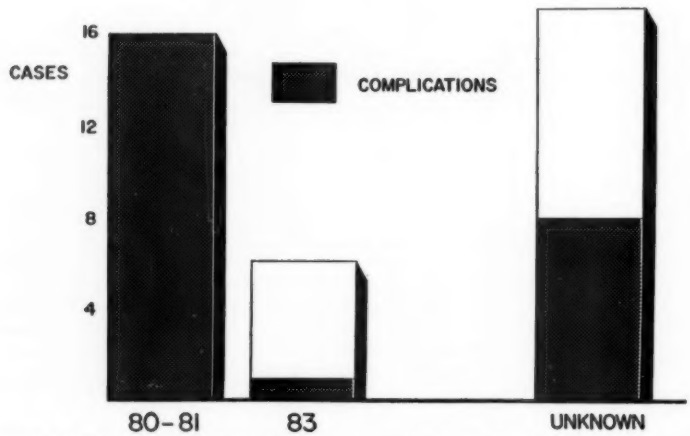

Fig. 44. Relationship of bacteriophage type to surgical complications of staphylococcal pneumonia. All 16 patients with 80–81 phage type had empyema.

pyopneumothorax or pneumatocele should make one strongly suspect that he is dealing with the 80–81 phage. By the same token, if an organism with this phage type is isolated from another source such as a subcutaneous abscess, the blood or a burn surface, a thoracentesis and thoracotomy set should be placed at the patient's bedside, a close record of the respiratory rate maintained on a continuous chart readily viewed, and frequent x-ray studies obtained of the chest so that one can deal adequately and quickly with pulmonary complications should they occur.

The choice of antimicrobial therapy may bear some relationship to the outcome of this disease. It seems clear that penicillin has little place in the management program, since in all but one patient the organism was insensitive to penicillin by standard sensitivity tests. In view of the excellent results recently reported by Perry et al.[5] in the management of severe staphylococcal infections in infancy, we recommend the use of intramuscular bacitracin in a daily dosage schedule of 1000 to 1500 units per kilogram in 4 divided doses for 4 to 5 days. After the short course of bacitracin a bacteriostatic drug to which the organism is sensitive, such as erythromycin (dose, 30 to 50 mg. per kilogram per day), should be given for at least 2 weeks after the temperature has been normal. Bacitracin is of particular value in infants less than one month of age. Ten of the 12 infants of this age in this series succumbed in spite of wide-range bacteriostatic antimicrobial programs in supportive therapy. Both the infant survivors received bacitracin.

Five of the infants less than 1 month of age who died received chloramphenicol in doses far exceeding 50 mg. per kilogram per day. Two infants died in a shocklike state at a time when their infection seemed under control. A similar fatal picture apparently due to the toxic effects of chloramphenicol has been previously reported[6] and may have played a part in these cases. If this agent is used in young infants, the daily dose should not exceed 50 mg. per kilogram per day.

SUMMARY

Thirty-nine cases of primary and secondary staphylococcal pneumonia have been reviewed. The over-all mortality rate was 46 per cent. In the 25 cases of primary pneumonia, surgical complications occurred in 19 (76 per cent). Empyema was the most common complication and occurred in 17 patients.

Late recognition and inadequate treatment of empyema, pyopneumothorax, lung abscess, acute pneumatocele and tension pneumothorax appeared to be responsible for or contributed to the death of six patients. By contrast, all patients in whom these surgical complications

were recognized and adequately treated, usually by closed thoracotomy, survived.

Conservative medical management alone, including needle aspirations of the pleural space in the presence of empyema or pneumothorax, is unwarranted.

All physicians should be prepared and equipped to establish closed thoracotomy drainage immediately after demonstration of the development of a pleural complication in a patient suspected of having staphylococcal pneumonia. The technique for the performance of effective closed thoracotomy is presented.

Attention is called to a previously unrecognized relationship between bacteriophage type 80–81 staphylococcal pneumonia and surgical complications which occurred in 100 per cent of these patients.

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CARDIAC LESIONS AMENABLE TO SURGERY

Current Status

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With the rapid changes and new developments in the field of cardiac surgery it is hard to keep abreast of the current trends. Surgical residents who trained in cardiac surgery a few years ago return to find that the operative procedures for many of the cardiac lesions have changed and that several conditions previously considered inoperable are now being corrected.

The operative procedures used in some cardiac lesions are well established, but in other conditions there is still a divergence of opinion as to the best means of surgical correction. The following is the current practice at the Children's Memorial Hospital.

PATENT DUCTUS ARTERIOSUS

The treatment of this condition has become well established with little change in technique for the past 10 years. Because of the low mortality, 1 death in a series of 500 cases, operation is advised for any patient in whom the diagnosis is established. The optimal time for operation seems to be about three years of age. Division of the ductus with suture of the stumps is to be preferred over ligation, though the latter procedure may have to be chosen in the case of an infant in critical condition.

Two complications of patent ductus arteriosus have stirred up the

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waters of an otherwise calm sea. One is the presence of pulmonary hypertension, and the other is the very young infant in cardiac failure. Pulmonary hypertension due to a large left-to-right shunt has caused no alarm, since the pulmonary artery pressure will drop dramatically as soon as the ductus is occluded. Hypertension due to increased vascular resistance, on the other hand, has been a difficult problem. Pressure recordings in the pulmonary artery are obtained during operation in these cases. If the pressure drops or even remains the same, closure of the ductus is advisable; however, if the pressure in the pulmonary artery rises after occlusion of the ductus, the operation should be terminated. Occlusion of the ductus in these cases closes off a release mechanism, and right-sided heart failure will result. Six patients with rise in pulmonary pressure after occlusion of the ductus have had division and suture of the patent ductus. Three have died, two are in heart failure, and only one patient is doing well at present.

Certain young infants with patent ductus pose a problem. Often the diastolic component of the murmur is absent, making diagnosis difficult. Retrograde aortography may be necessary to differentiate between ductus and interventricular septal defect. These patients may be in failure from birth and remain so until the patent ductus is closed. Several patients only a few weeks old have been operated upon with dramatic results.

COARCTATION OF THE AORTA

The treatment of this condition also has become fairly well fixed. Patients with adult type of coarctation with significant obstruction are advised to have resection of the coarctation and end-to-end anastomosis of the aorta. The optimal time for operation seems to be about 10 years of age, when the aorta has achieved fairly good size. The mortality increases with every decade thereafter.

Most infants with coarctation can be tided over with careful medical management until they are large enough to have an adequate lumen at the anastomosis.

There has been but 1 death in a series of 96 patients with resection of adult type of coarctation. This patient was an infant with fibroelastosis.

Several attempts have been made to correct infantile type of coarctation, using homologous vessel grafts or, in one instance, the subclavian artery to bypass the narrowed area. In every case the ductus was occluded, and death resulted from right-sided heart failure. Some method of gradual occlusion of the ductus must be used to allow the pulmonary vascular bed and right side of the heart a chance to adapt to the new situation.

VASCULAR RING

Little change in diagnostic measures or operative technique has been made since the original contribution by Gross. If the esophageal and tracheal compression is enough to produce distressing symptoms, the encircling vessel is divided at a point which will give maximal release of the structures. In the small infant with severe compression of the trachea the main difficulty is in maintaining an adequate airway during and immediately after operation.

TRICUSPID STENOSIS AND ATRESIA COMPLEX

There is nothing at present to offer patients with this condition except a shunting procedure. Complete intracardiac correction of this lesion would involve closure of the interatrial and interventricular septal defects, installation of an artificial tricuspid valve, enlargement of the diminutive right ventricle and correction of the pulmonary stenosis or atresia usually present. This is not yet feasible.

Most of the 82 patients coming to surgery were infants with extreme anoxia developing as the ductus arteriosus closes. An aortic-pulmonary anastomosis has been found the most satisfactory type of shunt and is often done in great haste, owing to the precarious condition of these tiny infants. Though the postoperative and late mortality is high, many patients are benefited greatly.

There is a question as to whether it is necessary to enlarge the interatrial septal defect or not. At present we are in the process of reviewing 36 pathologic specimens of operated and nonoperated cases in an effort to solve this problem.

ISOLATED PULMONARY STENOSIS

Great improvement in results of valvotomy has occurred since the adoption of direct-vision technique. Complete incision at the valve commissures is best obtained by opening the pulmonary artery after occlusion of the venae cavae and right and left pulmonary artery branches. This can be done with either hypothermia or extracorporeal circulation. We have chosen hypothermia with inflow occlusion, since it affords ample time to perform the valvotomy, which takes only two or three minutes. The additional personnel, expense and time consumed with extracorporeal circulation so far have not seemed warranted for such a brief procedure.

In 3 instances out of 42 patients undergoing direct-vision valvotomy, infundibular stenosis was found. In each instance there was adequate time to resect the obstruction. There has been no instance of unexpected interventricular septal defects found in this group. The possible

occurrence of these septal defects might eventually sway one toward extracorporeal circulation.

Patent foramen ovale frequently accompanies "pure" pulmonary stenosis, but it is doubtful whether anything active need be done about closing this defect, since diminution in size and pressure of the right atrium will allow for closure of the foramen.

Blind transventricular valvotomy was done on an earlier group of 89 patients. Eight have had enough recurrence of stenosis to require reoperation and direct-vision attack on the valve. In certain instances of tiny infants with severe obstruction, right-sided heart failure and general moribund appearance, a rapid transventricular valvotomy with a small knife will result in dramatic improvement on the operating table. Subsequently the expanding valvulotome and dilator can be used to complete the job. Several patients would have died except for this emergency procedure. Hypothermia or extracorporeal circulation seems out of the question in this type of case. Subsequent open procedure will probably be necessary at an older age.

Certain patients with a mild pulmonic valve stenosis may not require surgical relief. However, if the patient has symptoms of fatigue and dyspnea referable to the cardiac lesion, or has evidence of right-sided heart enlargement and strain, operation is advised. In the absence of this finding, but with a right ventricular pressure of 70 mm. of mercury or above, valvotomy is advised also. There has been only 1 death in the total of 131 valvotomies, showing that this procedure carries a relatively low risk.

OSTIUM SECUNDUM INTERATRIAL SEPTAL DEFECTS

There was a rapid shift from the closed, blind techniques devised for closure of interatrial septal defects to the open techniques, utilizing hypothermia or extracorporeal circulation. Again, as in the case of "pure" pulmonary stenosis, hypothermia affords adequate time to close a vast majority of ostium secundum defects successfully. If all findings, including catheterization data, suggest that the defect is a simple ostium secundum type, hypothermia with occlusion of venae cavae, pulmonary hili, aorta and pulmonary artery is used. Atrial exploration and closure of the defect with an over-and-over silk suture are possible in six minutes or less.

This procedure has been used with success in 22 cases. Two patients correctly diagnosed as having ostium primum defects were operated upon with hypothermia. Closure was attempted in one instance, resulting in death. The other patient was closed, to be operated upon later with extracorporeal circulation.

At present if there is any question as to whether the defect is ostium

primum type or has anomalous drainage of the pulmonary veins, the case is operated upon with extracorporeal circulation.

With such a low mortality for the operation, all patients with interatrial septal defects with any significant shunt are advised to have operative correction.

INTERVENTRICULAR SEPTAL DEFECTS

There is no doubt that these patients must be operated upon with extracorporeal circulation. The time required to do a thorough and careful closure with adequate exploration of the septum for other defects far exceeds the 10 minutes allowable with hypothermia.

A complete and lasting closure without interference with the conduction mechanism or valve leaflets is the goal of the operation, and good visibility and lack of pressure of time will aid in achieving it. For this reason cardiac arrest, either with potassium citrate or hypothermic coronary perfusion, would seem advisable. Careful placement of sutures is necessary in order to avoid the bundle of His. Multiple sutures of 5–0 silk with fine needles facilitate this objective. In larger defects an Ivalon patch would seem to permit closure without undue tension so that shallower bites of tissue are necessary.

Pulmonary hypertension is even a greater problem. In a series of 50 patients operated upon for closure of interventricular septal defects all 5 deaths were in patients in whom the pulmonary artery pressure almost equalled the systemic pressure. Even with the help of longitudinal sternal splitting incision, tracheotomy, positive pressure respiration and flap-valve patches, this group of patients remains a difficult problem.

The operative risk for patients without or with minimal pulmonary hypertension is so low that it would seem proper to advise closure of all interventricular septal defects that are hemodynamically significant.

OSTIUM PRIMUM INTERATRIAL SEPTAL DEFECTS

The time necessary for careful exploration of these defects, repair of the cleft mitral and tricuspid valves and meticulous suture of a patch over the defect so as to avoid the conduction mechanism renders use of extracorporeal circulation essential. The poor prognosis in this group of patients without operation makes successful closure even more gratifying. Of 10 patients undergoing surgery in this manner, all have done extremely well postoperatively and should have an excellent long-term outlook.

TETRALOGY OF FALLOT

There are many facets to the surgery of this condition that will require time alone to determine their worth. Obviously, complete correction

of the cardac lesion is the ideal goal, but operative mortality must also be kept in mind.

Closure of the interventricular septal defect and opening up of the outflow tract to the lungs must be done with extracorporeal circulation. It is the type and position of this outflow obstruction and the size of the subsequent pulmonary artery that affect the operative risk and long-term prognosis.

Valvular stenosis and easily resected infundibular obstruction with a sizable pulmonary artery to empty into are the most favorable cases. If a patch of Ivalon or other material must be sutured to the ventricle to allow for enlargement of the outflow tract, the risk is greater, and the eventual fate of the prosthesis with growth is unknown. If the patch must extend into the pulmonary artery to enlarge the pulmonary valve ring, the mortality rises sharply, and the long-term outlook is even more in doubt. In a group of 20 patients operated upon with extracorporeal circulation 4 of the 5 deaths were in patients who had to have patches sewn to the outflow tract.

At present the policy is to advise extracorporeal circulation and correction of tetralogy defects for patients who have evidence of a fair-sized pulmonary artery by selective angiocardiogram. On the other hand, the very blue infant with no murmur or the patient who has a small pulmonary artery demonstrated by angiogram is operated upon, doing either an aortic-pulmonary or subclavian-pulmonary anastomosis.

Preliminary impressions of a detailed study of patients who underwent aortic-pulmonary anastomosis 12 or 13 years ago suggest that the ones that did best were the children with small pulmonary arteries. Time alone will tell what place, if any, the shunting procedures will occupy in the treatment of tetralogy of Fallot.

AORTIC STENOSIS

After trying blind transventricular and transaortic valvotomies and direct-vision approach under hypothermia, I am sure the safest method is to use extracorporeal circulation without cardiac arrest, but with coronary artery perfusion. The strained and hypertrophied left ventricle takes poorly to anoxia and is difficult to bring out of cardiac arrest or fibrillation. This is far different from the right ventricular strain and hypertrophy as seen in isolated pulmonary stenosis.

The congenitally deformed valve may be difficult to convert to normal function, since the leaflets may be twisted and deformed, with the consistency of cartilage. The production of regurgitation is to be avoided, since it may be as disabling as the original lesion.

Subvalvular stenosis, if resectable to a sufficient degree, may give excellent results, since the valve itself may be completely normal.

Patients with aortic stenosis with symptoms of dyspnea and fatigue or passing out spells, with significant evidence of left-sided heart strain or with a hemodynamically important gradient across the valve are candidates for valvotomy. Many of these apparently healthy children may die suddenly at play or at rest.

ATRIOVENTRICULAR CANAL

This lesion has many variants. The atrial component may be large, and the ventricular defect small. In such cases closure with a plastic patch may be feasible and fairly safe. If, however, the ventricular septal defect component is large, closure of the opening may be difficult, and interference with valve function and conduction mechanism is likely. In such cases the operative risk is almost prohibitive.

TRANSPOSITION OF THE GREAT VESSELS

As evidenced by the many and varied attempts at surgical treatment of this condition during the past decade, the problem is difficult and as yet incompletely solved. Most recent surgical procedures have been aimed at reversing the venous inflow of the heart rather than the great vessels, since the problem of the origin of the coronary arteries seems insoluble.

A series of 130 patients have been operated upon by switching the inferior venacaval flow via an aortic arch graft to the left atrial stump of the divided right pulmonary veins and anastomosis of these pulmonary veins to the right atrium. Though this is an incomplete correction, since the superior caval and left pulmonary vein flow remains unreversed and associated lesions are not corrected, the operation is to date the best that can be offered the patient. The mortality of 30 per cent seems high, but many of these otherwise doomed patients benefit greatly by the procedure.

Work is progressing on more complete correction by means of extracorporeal circulation and intracardiac plastic procedures on the interatrial septum and pulmonary veins. No conclusions can be reached about these attempts until more experience is available.

AORTIC-PULMONARY WINDOW

This lesion usually produces enough strain on the heart to make closure imperative. Diagnostically, this may simulate a patent ductus or a high interventricular septal defect with prolapse of an aortic valve cusp. Most attempts at closure of the defect have been done in a manner similar to suture or division and suture of a patent ductus. In a recent

case a huge aortic-pulmonary window was discovered in a patient operated upon as having a possible high ventricular defect with diastolic reflux due to prolapse of the aortic valve into the defect. Since the patient was prepared for extracorporeal circulation, he was put on "by pass," and the task of division and suture of the defect was rendered safe and simple.

ANEURYSM OR RUPTURE OF THE SINUS OF VALSALVA

The best method to repair such a defect is by means of extracorporeal circulation. The defective aortic valve cusp can be repaired, and the aneurysmal tract and its entry into one of the chambers of the heart or great vessels can be obliterated. One case in which the aneurysm ruptured into the left ventricle was operated upon with successful closure of the defect; however, the huge left ventricle could never be forced into a potent beat even though cardiac arrest had been brief. Instances of rupture into the right ventricle have been operated upon with success. This is another example of the intolerance of the hypertrophied left ventricle to anoxia.

Certain cardiac lesions seem not to be amendable to surgery at present. Single ventricle, truncus arteriosus and hypoplasia of the aortic tract complex are examples. With the rapid advances in the field of congenital cardiac disease combining contributions by pathologist, diagnostician and surgeon, more and more lesions will become remediable, and the operative treatment will improve steadily.

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JAUNDICE

The Surgeon's Role in Diagnosis and Management

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The problem of jaundice in childhood is one that encompasses the skills and knowledge of all aspects of the care of children. Those who are primarily interested in the medical care as well as those interested in the surgical care of children become deeply involved, since the differential diagnosis of jaundice in childhood is indeed a most complex matter. The discussion of childhood jaundice is best divided into jaundice of the neonatal period; secondly, jaundice of young infancy, with the dividing line perhaps at six months of age; and, thirdly, jaundice of older children. The matter of differential diagnosis of jaundice in these three periods and the surgeon's role in such a problem will be presented herewith.

JAUNDICE IN THE NEONATAL PERIOD

Jaundice in the immediate neonatal period, that is, the first week in life, is almost invariably associated with hemolytic disease. The etiologic factors behind this hemolytic disease are either blood group incompatibility or, more commonly, RH incompatibility. In addition to these two etiologic agents, severe sepsis in the newborn will cause hemolysis and jaundice, and other evidence of infection such as syphilis must be considered. It is obvious in all these that the role of the surgeon is most menial. The only concern the surgeon might have for these diseases mentioned might be assisting with the mechanics of exchange transfusion should the umbilical vein not be available. Saphenous cut-down with intubation of the external iliac or vena cava is a most acceptable method. Many nonsurgeons doing the exchange

transfusions prefer surgical help in isolating these vessels and caring for the subsequent wound. Aside from such an aid and assistance, a surgeon is rarely involved in the problem of jaundice in the first week of life.

JAUNDICE IN INFANCY

The next group of patients is the one in which the surgeon is most intimately involved and must work hand in glove with the family physician or pediatrician. The principal role of the surgeon in the jaundice in this group is to diagnose and treat the children afflicted with atresia of the bile ducts or other mechanical obstructions. These conditions are obviously incompatible with life and must be remedied reasonably early in the patient's life so that irreversible damage from obstruction is not done. Numerous attempts have been made to decide the "point of no return" as far as obstructive jaundice is concerned. As young as two and somewhat more commonly three months of age, infants have been observed to have moderately advanced cirrhosis due to obstruction which has led to portal hypertension, esophageal varices, ascites, liver failure, or hemorrhage and death. Such a course of events in these tiny infants makes it evident that definitive therapy for atresia of the bile ducts must be carried out as early as possible, preferably about two months of age and certainly before three months of age.

The ability to make a differential diagnosis at this very early age has taxed and does tax the ingenuity of all concerned. In the 1930's Ladd[6] mentioned infants who were suspected of having total biliary obstruction, but who at operation had an intact external biliary tree. Coincident with opening of the bile ducts by flushing the system with saline solution, many of these children began to function normally and recovered from the jaundice. To this group of patients he attached the title of "inspissated bile" syndrome or biliary plugs. Since Dr. Ladd's time the terminology has continued, and people have referred in the literature to this vague entity of biliary obstruction due to highly viscous or inspissated bile. It became evident that many of these cases would clear up without surgery and equally evident that anesthesia and attendant surgical trauma with a complete biliary exploration were most harmful to many of these children and undoubtedly caused the demise of some, adding the insult of anesthesia to an already damaged liver.

In the last decade the concept of "inspissated bile" has been added to or in some quarters replaced by the diagnosis of neonatal jaundice due to hepatitis. It is assumed that the virus of hepatitis can cross the placental barrier, and that, though the mother has subclinical infection, the baby may have obvious clinical jaundice and clinical disease.

In the severe form of hepatitis of the infant total biliary obstruction may occur. It is in these children that the differential diagnosis between atresia or mechanical obstruction of the bile duct and obstruction due to diffuse parenchymal disease arises. In 1952 Gellis et al.[3] reviewed the children operated upon because of biliary obstruction and found to have the so-called inspissated bile syndrome or parenchymal disease causing the jaundice, and came to the conclusion that surgery in this group of patients with the attendant complete biliary exploration necessary to reveal an intact biliary tree was the cause of the high morbidity and mortality in the series reviewed at the Boston Children's Hospital for the decade prior to their writing. With this series of cases as a base these authors pointed out the low salvage rate for biliary atresia, which in the best of hands is between 10 and 20 per cent. They felt that the high degree of morbidity and mortality associated with anesthesia and exploration in this group of patients mandated that operation not be carried out before five or six months of age, allowing those who would clear spontaneously to do so and those who had undoubted atresias to manifest themselves by progressive evidence of obstruction.

Needless to say, particularly from the surgeon's standpoint, this concept was not greeted with universal approval. Waiting until five to six months of age was practically assuring long-standing liver damage and a fatal outcome even to the correctible case of biliary atresia. In response to this suggestion of "the waiting game" various authors, including Swenson[7] and Koop[5] among others, have indicated the more expedient and briefer surgical procedure of liver biopsy and cholangiography to determine the patients that are surgical candidates as opposed to those who have medical disease. Basically, their method is dependent on the use of operative cholangiography and experienced anesthetic support as well as experienced pathologic judgment in the appraisal of liver biopsy. With a moderate incision the porta hepatis can be exposed, and should a gallbladder be present, a small polyethylene tube can be sewed in place; after a suitable irrigation of the biliary tree contrast medium can be injected and films obtained. Figures 45 through 48 are examples of the information that can be gained from such a study.

Figure 45 shows gallbladder, cystic duct and common duct, but fails to show the branches of the hepatic ducts. At exploration it was evident that this child had an intact tree, though perhaps hypoplastic, since there was a small amount of bile in the gallbladder. The entire procedure on this child took about 25 minutes. She showed no immediate or long-lasting ill effects from operation and gradually began to lose her jaundice and cleared spontaneously. Liver biopsy showed no evidence of hepatitis, and it was felt that this patient had a hypoplastic but patent biliary tract.

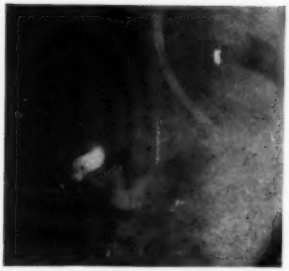

Fig. 45. Normal cholangiogram without filling of the hepatic ducts.

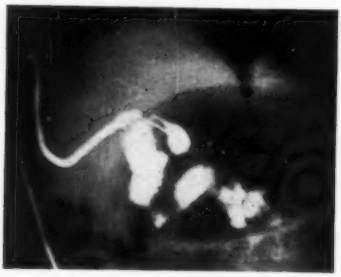

Fig. 46. Operative cholangiogram showing atretic hepatic ducts.

Fig. 47. Operative cholangiogram showing dilatation and obstruction of common duct.

Fig. 48. Postoperative cholangiogram showing free flow into duodenum.

Figure 46 shows the presence of the gallbladder, cystic duct and common duct in communication with the duodenum, but no evidence of hepatic radicals. This gallbladder was empty of any bile and contained just a small amount of mucus, and detailed exploration indicated that the atresia was in the hepatic ducts. No remnant of these ducts could be found, and this child was necessarily deemed uncorrectible.

Figure 47 shows the gallbladder and hepatic radicals in addition to the common duct with dilatation of the distal common duct, but no communication with the gastrointestinal tract. The contrast material seen outside the biliary tree is extravasation from the point of injection. It was possible to anastomose this distal common duct to the duodenum, and Figure 48 shows a cholangiogram taken 2 weeks later with rapid passage of the dye from the gallbladder into the duodenum without evidence of obstruction.

These examples, then, show the use of operative cholangiography pointing the way to more complete surgical exploration.

There is some difference of opinion as to the interpretation of the liver biopsy. Some have advocated that this be done as a frozen section and the presence or absence of hepatitis be determined by this method. Most feel, however, that this is expecting too much of the pathologist under the rapid section technique and that should adequate doubt be present as to the presence or absence of parenchymal disease, the procedure should be terminated and the decision based on permanent sections.

This most obviously brings up the matter of how to obtain a section of liver without surgical intervention in order to prevent even this short surgical exposure in a patient who has parenchymal liver disease. There have been champions from time to time of liver biopsy by the aspiration method. A most recent compilation of this method and evaluation comes from Kaye et al.,[4] who give their experience in needle biopsy of the liver as carried out in 25 infants age 3 weeks to 18 months. These authors point out the strict criteria for the use of this method, indicating that normal bleeding, coagulation and prothrombin time must be obtained in these patients, and that most careful technique in obtaining the biopsy must be followed as described by them. This must not be done in the absence of surgical assistance should abnormal bleeding be encountered. There is also a serious problem in the matter of interpretation of the specimen and the adequacy of the specimen obtained. These authors point out the number of times that secondary aspiration had to be carried out and also the equivocal results obtained in some of the cases. This small series points out the dangers inherent in broad advocacy of aspiration needle biopsy, since the variables present are many. There is no doubt of the fact that this

is another diagnostic tool which may help in the most severe problem case, but cannot be said to be the panacea for all problems in differential diagnosis and cannot be advocated or done without certain risks. Certainly this method should not be undertaken in the absence of maximum support from the surgical, pathologic and laboratory teams of the highest caliber familiar with the problems of the neonatal period.

Since the differential diagnosis of jaundice in this period seems to be a matter of surgical interest, how, then, can the surgeon or medical compatriot turn to the laboratory and expect some aid? Early in the discussion of neonatal jaundice some authors advocated depending on progressive elevation of the serum bilirubin, taken in serial determinations, to be positive of mechanical obstruction. It is evident to many and now accepted as more valid fact that the serum bilirubin, even in the face of complete obstruction, may initially peak and then drop to a plateau and not climb until far-advanced liver damage has been incurred. Obviously, if one waits until this point for a definitive diagnosis of atresia, irreversible changes have taken place and surgical correction will be to no avail; thus the serum bilirubin does not give any great amount of help in the differential diagnosis, other than to indicate obstruction from some cause.

It is necessary, however, to view the serum bilirubin in light of recent knowledge. Waters[8] and others have clarified thinking in this matter by showing that the so-called direct bilirubin, determined by the immediate phase of the diazo reaction, is in actuality measurement of bilirubin conjugated with glucuronide to form bilirubin glucuronide. This conjugation takes place in the liver with the interaction of the enzyme transferase.

The so-called indirect or delayed phase of the diazo reaction is a measurement of the unconjugated bilirubin, when the conjugated bilirubin has been subtracted from this total.

With this concept an elevated conjugated bilirubin is a manifestation of inability of the liver to secrete the bilirubin, when conjugated, owing either to an obstruction in the extrahepatic biliary tree or parenchymal damage due to liver disease. In either case it becomes obvious that a high conjugated bilirubin is not a specific test for extrahepatic obstruction such as atresia of the bile ducts.

A high value of the unconjugated bilirubin may then manifest excessive bilirubin due to hemolysis and/or the inability of the liver to conjugate normal amounts of bilirubin. Parenchymal liver disease may thus show either elevated conjugated or unconjugated bilirubin, or both. The serum bilirubin test is thus not differentiating.

The addition of the other liver function tests, such as measurement of turbidity, flocculation or transaminase activities, may or may not shed

light on the problem. Early in the course of jaundice, elevation of any of these values may point to parenchymal disease on an infectious basis rather than pure obstruction. As the duration of the jaundice is prolonged, high values of these tests simply show liver damage, but do not help in determining the etiology.

It is obvious, therefore, that one must establish a program of treatment for patients with jaundice in the first month of life. No one set of criteria can be used to diagnose the jaundice of a mechanical obstruction which must be operated upon early to achieve the salvage rate, low though it may be, that is possible under the present circumstances. Most jaundice of the newborn period is not discovered or not evident enough to warrant the attention of the physician until three to four weeks of age, and often not until six weeks of age. Once it is discovered and proved by laboratory methods to be significant jaundice, it behooves one to make a careful study of this child to differentiate between the patient with atresia or mechanical obstruction who may be helped surgically and the child with medical disease who in all likelihood will clear up with expectant medical care. A careful history and suitable laboratory tests should be used to rule out hemolytic disease based on congenital spherocytosis or sepsis.

Cytomegalic inclusion disease as a cause of jaundice must be kept in mind. Rare though this may be, a qualified pathologist can recognize the inclusion bodies in the sediment of the urine and thus aid in the diagnosis.

Baseline liver function tests are important from the standpoint of the status of the patient at the time of the initiation of investigations. Stool and urine studies for the presence of bile and urobilinogen are important, though somewhat misleading at times. Yellow stools have been reported frequently in patients with complete obstruction; these may be due either to staining from a highly concentrated urine or to staining of the stool from bile-stained succus entericus, saliva and other fluids transmitted through the bowel wall. Though in the face of complete obstruction one would expect a negative stool and urine urobilinogen, this substance may be found in small amounts even in the obstructed patient. Most investigators have discarded this as an absolute test for the presence or absence of obstruction.

It has been most helpful in many cases to do a duodenal intubation to determine the presence or absence of bile in the duodenal contents. This may be done under fluoroscopic control and, by the more experienced practitioner, without the aid of fluoroscopic control, obtaining a sample of duodenal contents distal to the ampulla of Vater. If biliary excretion into the intestinal tract is beginning to be re-established, minute amounts of bile can be determined chemically in this way, indicating reasons for delay of operation. Completely

negative samples taken from the duodenum potentiate the decision for surgical intervention.

In the very young patient it is justified to use a period of observation with or without the use of hydrocholeretic agents such as Decholin or Ketochol. Such agents have been proposed by Gross[1] and others in this waiting period to clear the biliary tree of inspissated bile. Others have felt that perhaps such agents have caused increasing damage in the face of an already damaged liver with parenchymal disease and have not been enthusiastic about their use. This author is in favor of using the hydrocholeretic agents for a short period of time in the young patient, but not to the exclusion or delay of operation in the patient who is approaching three months of age. After a course of watchful waiting with or without the hydrocholeretic agents, if the laboratory studies remain the same and there is no evidence of a decrease in serum bilirubin or clinical appearance of lessened jaundice or laboratory evidence of bile in the stool, surgical exploration as outlined should be carried out. By using this method in more than 30 babies in the past 6 years this author has evidenced no case of postoperative mortality and no severe morbidity directly attributable to the anesthetic procedure. Only one child was operated upon (Fig. 45) who had an intact biliary tree that was found at the time of operation. Two other children, in whom no normal biliary tree could be found at operation, later showed clearing of the jaundice; one of them proved to have an accessory bile duct not in the usual position at postmortem examination following death from cirrhosis. The other is still alive and without jaundice.

As to the surgical management of the jaundiced patient after the problem of differential diagnosis has been answered, the surgical technique used in alleviation of the jaundiced patient with atresia must of necessity be dictated by the anatomic condition present. Ladd[6] and Gross[1] and others have advocated direct suture of the duodenum to the remnant of the biliary tree wherever it may be. They have had reasonable success with this technical procedure. Because some of these patients had repeated ascending cholangitis, Koop and Kiesewetter[5] and others felt that a Roux-en-Y procedure with anastomosis of the end of the jejunum to the remnant of duct would be safer from the standpoint of repeated infection. This author believes that the direct anastomosis is probably more valid, shorter, and technically more feasible in most cases. The presence or absence of ascending cholangitis is a function of the adequacy of the anastomosis and the lack of obstruction at the anastomotic site rather than the presence of intestinal contents and stomach contents across the anastomotic site.

By the time the remnant of biliary tree is discovered after prolonged investigation, an even more prolonged anastomotic technique would

be injurious to the child. Again this author has found that in several cases it has been advantageous to establish an external biliary fistula once a remnant has been found at the end of a long procedure and allow this child, through biliary tract drainage by catheter, to recover from the anesthetic procedure, to lose the jaundice, and to be built up somewhat, and have re-exploration in two to three weeks with anastomosis at this time under more favorable conditions. One patient in particular was salvaged by this method who was in poor condition at the time the remnant of hepatic duct was discovered during exploration. Other technical details are a matter of individual preference of the surgeon. Obviously the principles of careful anesthetic management, adequate preoperative preparation, blood replacement, and general familiarity with the problems of infancy are important in the surgical approach to these obstructed patients.

JAUNDICE IN OLDER CHILDREN

The third group of patients that we should consider are those who have obstructive jaundice in the period following the first six months of life. In the earlier period we were concerned with the differential diagnosis between atresia of the bile ducts and parenchymal disease. In the older children we are concerned with other forms of acquired biliary obstruction, though some patients with congenital defects can be affected later in life. Idiopathic dilatation of the common duct, otherwise known as choledochal cyst, is a condition often associated with intermittent jaundice and intermittent evidence of obstruction, occasionally discernible by palpable abdominal mass, but more often no mass is palpable. The bile ducts are patent, but there is a large cystic dilatation of the distal duct with a small ampulla of Vater. This evidently intermittently obstructs and causes progressive damage. Anastomosis of the duodenum to this cystic dilatation is the simplest solution to this. Attempts at removal of the cystic dilatation of the duct have been met with a larger operation than is necessary and often irreparable damage to adjacent structures.

It would be impossible to discuss all other forms of obstructive jaundice in the older children. Tumors may arise within the liver or in the porta hepatis and cause obstruction to the external hepatic tree. Lymph node enlargement associated with reticuloendothelial disease may likewise obstruct the ducts. Long-standing liver damage associated with Banti's disease or portal hypertension from other causes may also be associated with late jaundice. The surgeon's role in these will be dictated by the condition of the patient and the coexistence of other forms of disease. The problems of jaundice in the older children are more to be considered with adult criteria in mind. Gallstones and

biliary obstruction therefrom are uncommon in children, but must be looked for in association with severe forms of hemolytic disease and should be considered a possibility in biliary obstruction in any age group, particularly if there has been a history of some hemolysis or hemolytic disease of a congenital or acquired form.

SUMMARY

In summary, one can say that the problem of jaundice in the first three months of life is one that must be faced with the teamwork of the medical and surgical approaches and can be extremely complex. The exact method of handling the jaundiced child and determining whether he is obstructed from a mechanical developmental defect or from parenchymal disease will depend on the facilities present. Liver biopsy, preferably by the open method with accompanying cholangiogram, is the preferred method; aspiration biopsy controlled carefully and in expert hands is a helpful adjunct. There is no substiute for a carefully taken history, adequate laboratory examination and cautiously arrived at decision for surgical intervention to prevent the patient with correctible atresia from having irreversible liver damage due to prolonged obstruction. Surgical intervention can take place in the child with parenchymal disease if such is necessary to make a differential diagnosis without severe and dire effects on the patient.

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ABDOMINAL PAIN IN CHILDREN

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The study of abdominal pain in infancy and childhood leads to an understanding of many conditions requiring the care of the general practitioner, the pediatrician and the pediatric surgeon. Clinical acumen rests upon a thorough grounding in fundamentals, and there is no substitute for a painstaking history and physical examination, despite the plethora of radiologic and laboratory aids to diagnosis.

THE HISTORY

Children younger than three years can give no history, and we must look to the parent. As a rule it is well to take the history in the presence of the child so that he may hear what is said and may become accustomed to the physician, who, in turn, can observe the child. This also gives the physician an opportunity to allay the child's fear, inspire confidence and create mutual friendliness. Older children can usually give a satisfactory history, often more reliable than that given by the parent, particularly when the child is over seven or eight years of age. The physician should take into consideration the mental and emotional development of the child and the parent. The overprotective parent often tries to dominate the interview. When the parent is emotionally disturbed, he or she is taken to another room and questioned in a calm, unhurried manner.

Disease of the gastrointestinal and the genitourinary tracts is the cause of most abdominal pain, which is explained on the basis of either distention of the walls of the viscera, or changes within the tissues

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resulting from inflammation. In inflammations of the gastrointestinal tract there is usually spread of infection to the parietal peritoneum, and the inflamed peritoneum also gives rise to abdominal pain.

An analysis of abdominal pain concerns first the *site*, which is useful to determine the abdominal organ from which it is derived. The abdominal viscera are innervated by sensory afferent fibers of the sympathetic chain which pass to the dorsal root ganglia between the fifth thoracic and third lumbar cord levels. Midepigastric pain may arise in the stomach, duodenum, pancreas, biliary tract or liver. Periumbilical pain may arise in the small intestine, appendix, upper ureters, and testis or ovary. Hypogastric pain may arise in the colon, lower ureters, bladder, or pelvic viscera in the female. Thus, when pain arises in the abdominal viscera, it is first felt in the midline anteriorly. In inflammatory lesions with involvement of the parietal peritoneum the pain usually becomes more severe and better localized to the anatomic region of the organ. For example, in the early stages of acute appendicitis the pain is of visceral origin and located in the epigastrium. As the parietal peritoneum is involved, the pain becomes more severe and is located over the site of the appendix. The pain arising in retroperitoneal viscera such as the kidney and pancreas is often felt close to the site of the lesion. Through stimulation of the lower thoracic cord segments, pulmonary, pleural, cardiac, pericardial or mediastinal diseases may give rise to abdominal pain.

The second characteristic of abdominal pain to be considered is the *quality* and *timing*—aching, colicky, continuous or intermittent. The typical colic is characteristic of early intestinal obstruction. It comes and goes, and is due, in all probability, to intermittent peristaltic effects upon the part of the intestine to overcome resistance at the point of blockage of the bowel. This is well illustrated in the baby with intussusception, who will suddenly begin to cry and draw up his legs, repeating this episode at intervals, and relaxing at other times. As the lesion progresses the colicky pain is replaced by a steady pain due to the onset of vascular changes in the bowel wall.

A third characteristic of abdominal pain is *radiation* to other sites. This is illustrated by the pain of renal colic, which usually begins in the upper outer loin or in the costovertebral angle, and then radiates downward to the lower loin, groin, bladder, urethra, spermatic cord, testicle or inner thigh.

The final and important characteristic of abdominal pain is its *association with other symptoms*. A comprehensive summation of all symptoms must be obtained. The most common of these are nausea and vomiting. These are complicated processes brought about by the sympathetic and parasympathetic nerve supplies of the abdominal organs producing viscerogenic reflexes. In cases of intestinal obstruction

they are more directly related to the blockage of the intestinal tract, and characteristically the vomitus is bile- or fecal-stained and associated with distention of the abdomen and constipation. The importance of the history is shown in cases of pneumonia and in urinary obstruction associated with abdominal pain. In the latter the gastrointestinal manifestations may completely overshadow those of the urinary tract, and in any child with abdominal pain the presence or absence of urinary frequency, dysuria or hematuria should be elicited. The respiratory symptoms of a pleural or pneumonic infection may also be overlooked. Blunders in which a needless abdominal operation is performed when the disease is actually present in the urinary tract or chest still occur with relative frequency.

THE PHYSICAL EXAMINATION

Children are usually fearful of an examination, and the physician should try to allay this fear. Conversation about childhood interests, and playing with toys are often helpful. The physical examination of the infant or child whose chief complaint is abdominal pain should be unhurried and follow a plan of procedure, in the order described below. The clinical application of the various parts of the examination will be noted.

Inspection

The infant manifests abdominal pain by crying, irritability or refusal to eat. The child above the infant stage shows by his facial expression that he has pain. The general appearance gives the first indication of the severity of the child's illness, and the degree of shock or dehydration is often apparent. Weakness and pallor suggest hemorrhage. The attitude of the child is important. In severe pain or hemorrhage he may be restless. In conditions causing peritoneal irritation he usually remains quiet. The respiratory motion of the abdominal wall is observed on normal respiration and after taking a deep breath. Limitation of motion may indicate rigidity of the abdominal muscles. If abdominal distention is seen, its extent is observed—generalized or localized; if the latter, the area involved. The possibility of intestinal obstruction or an abdominal tumor is brought to mind. Intestinal peristalsis is often visible in intestinal obstruction, proceeding in a wavelike movement across the abdominal wall. This is more easily noted in infants, in whom the abdominal wall is thinner. Discoloration of the skin of the abdominal wall may be evidence of trauma, and hemorrhagic spots may be seen in blood dyscrasias. The presence or absence of abdominal scars is noted. The inguinal regions should be carefully observed for evidence of hernia.

Auscultation

The examiner should have a clear conception of the sounds of normal peristalsis. Peristalsis may be increased, diminished or absent. Absent peristalsis is often associated with paralytic ileus. Increased peristalsis may be found in 2 forms: (1) fairly constant, loud gurgling sounds which vary in intensity, but have no definite rhythm. Variations of intensity occur without any alteration of the patient's abdominal discomfort. Such sounds are heard in acute gastroenteritis or digestive upsets. (2) The sounds produced by the rhythmic contractures of the intestine in acute mechanical obstruction are characteristic. Loud, high-pitched sounds of gradually increasing intensity are heard; these rise to a crescendo and then gradually die away until only a few faint tinkles are heard. The patient is aware of painful cramps which increase and subside with the peristaltic activity. The abdomen is silent between episodes of pain.

Palpation

As a preliminary step, the older child should be asked to cough. In the presence of acute peritoneal inflammation the cough often elicits sharp pain in the involved area and localizes the area of inflammation. Palpation of the abdomen starts with the areas at a distance from this site. For example, if the "cough tenderness sign" is in the right lower quadrant, palpation proceeds from the left lower quadrant, to the left upper quadrant, to the right upper quadrant, and finally to the right lower quadrant. A valuable procedure is to have the child palpate his own abdomen in that sequence. This gives him a sense of participation in the examination, and he does it without fear of the examiner. Palpation should be performed gently with the warmed flexor surfaces of the fingers. Poking at the abdomen with the finger tips may produce false tenderness. The patient lies in a relaxed position, and is instructed to breathe deeply and quietly through the mouth. The degree and site of tenderness are noted.

Spasm of the abdominal muscles is produced by complex reflex mechanisms, and is a valuable sign of peritoneal irritation. The source of the peritoneal irritation may be inflammatory, chemical, traumatic or vascular. Segmental spasm of one rectus muscle (spasm limited to one quadrant) is encountered in early peritonitis. Extensive rigidity of both rectus muscles indicates a diffuse peritoneal irritation. At times it is difficult to differentiate between voluntary and involuntary muscle spasm. In such cases it is advisable to administer a mild sedative. Rigidity of the muscles of the loin is caused by renal or ureteral diseases, but may be produced by an inflamed appendix, retroperitoneal

in position. When the inflamed organ lies directly over the psoas muscle, there may be resultant flexion of the thigh. In eliciting the "psoas test" the patient may lie face downward and the thigh be then lifted upward, or the patient may lie on the side opposite to the one being tested, as the lower extremity is slowly forced backward.

Some of the muscles having reflex associations with intra-abdominal organs are situated at a considerable distance from the abdominal cavity itself. Disorders of pulmonary, pleural, cardiac, pericardial or mediastinal origin may irritate the abdominal nerves in the thoracic part of their course. In so doing, the thoracic disease will set up the same reflex phenomena as though the disease were actually located below the diaphragm. Examination of the abdomen without regard to the history or examination of other regions of the body might reveal a "surgical abdomen," when all too frequently an operation upon the "surgical abdomen" reveals no lesion, while the subsequent course and the accumulation of more complete data disclose that the actual seat of the disease was within the thorax.

The attempt to induce so-called rebound tenderness is a practice we condemn. The sudden release of the abdominal wall causes the child unnecessary pain, may be "positive" in the absence of peritonitis, adds no information of importance, and conceivably spreads infection.

Finally, some abdominal tumors are associated with abdominal pain, and a careful palpation of the abdomen will reveal many of them. Masses of hard feces are often palpable in the child with severe constipation.

Percussion

Percussion of the abdomen is useful for study of the costovertebral angles and tenderness over the kidneys, looking for shifting dullness in the flanks, and mapping out masses, or solid viscera as the liver and spleen.

Rectal Examination

This procedure should be performed in any child with abdominal pain, at the end of the physical examination. It is important to be both gentle and deliberate when performing a rectal examination. At best, children are frightened by this probing into their nether regions, and their cooperation can be obtained by explaining beforehand what the examiner is going to do. By ignoring these instructions the examination will be a harrowing and painful experience to the child. The patient is warned not to strain, but to lie quietly, breathing with the mouth open, and is informed as to exactly when the examination is

about to begin. The finger (index in the child, fifth in the infant) is held firmly against the anus until the sphincter is felt to yield, rather than thrust forcefully into the rectum. The finger, gloved and well lubricated throughout its length, is introduced slowly. The presence of hardened feces renders the examination unsatisfactory, and when such is encountered, the procedure is best deferred until a low enema or a suppository is administered.

There are often differences of interpretation between various examiners in the finding of tenderness on rectal examination. This observation in infants cannot be relied upon, and is of value only in examination of the older child. On the other hand, the finding of a mass or induration is a positive sign; the former may be an abscess or tumor, the latter, pelvic peritonitis. After having completed the examination the finger is examined for blood, mucus or pus. The rectal examination is usually the only means of diagnosing pelvic appendicitis, the abdominal examination in these cases being negative.

We feel that if the physician takes a complete history and conducts a careful physical examination (including the entire body) along the lines outlined above, the cause of abdominal pain can usually be ascertained. Recordings of temperature, pulse, blood cell count and urinalysis are routine accompaniments. Radiologic examinations of the chest and abdomen are indicated if there is a suspicion of pneumonia, genitourinary disease, abdominal tumor or intestinal obstruction. Gastrointestinal and barium enema examinations and excretory urograms are additional important diagnostic aids. Proctoscopy, sigmoidoscopy and stool examinations are necessary in the diagnosis of diseases of the colon or rectum.

SURGICAL DISEASES OF THE ABDOMEN

In children with acute abdominal disorders it is our opinion that when abdominal pain is accompanied by signs of peritoneal irritation—tenderness and spasm—it is safer to perform a laparotomy, unless a specific nonsurgical illness becomes quickly apparent on routine clinical and radiologic studies.

The most common abdominal surgical condition in childhood is *acute appendicitis*. There seems to be little diminution in the duration of symptoms and the incidence of peritoneal complications over the years. This is particularly true in infants, and in cases in which the appendix has an atypical location—retrocecal, pelvic, postileal or subhepatic. When the clinical picture is not well defined, it is important to examine the child at frequent intervals. If the child is febrile and

dehydrated, it is best to hospitalize him, so that he can be observed more easily and the necessary parenteral fluids administered. We decry the use of antibiotics prior to diagnosis. This may ameliorate the signs and symptoms, thus confusing the clinical picture, does not ensure control of the infection, and leads to a false sense of security in the face of a spreading infection. Any child with the combination of abdominal pain, vomiting, and tenderness in the region of the appendix should be considered to have acute appendicitis unless proved otherwise. One cannot rely on the temperature and white blood cell count; these may or may not be increased. The time to make a diagnosis of acute appendicitis is before the onset of peritoneal complications. A diagnosis after the appearance of peritonitis or abscess is usually an indication that the physician has not given enough consideration to the early symptoms and signs.

Acute mesenteric lymphadenitis is the most common condition which simulates the symptoms and signs of acute appendicitis in childhood. Many of these children have a history of prior attacks of abdominal pain, and have an infection of the upper respiratory tract. The pain is usually intermittent, and vomiting is less frequent than in acute appendicitis. Tenderness in the right lower quadrant of the abdomen is common to both conditions, but muscle spasm is much less frequent in acute mesenteric lymphadenitis. However, since mesenteric lymphadenitis may closely mimic acute appendicitis, operation is often indicated to avoid overlooking an acutely inflamed appendix.

Primary peritonitis (see also p. 967) due to pneumococcal or streptococcal infections has had a diminishing incidence in recent years, but can be confused with cases of diffuse peritonitis secondary to appendicitis or a ruptured viscus. An upper respiratory tract infection precedes or accompanies the peritonitis in many cases. Vomiting and diarrhea are common. These patients appear acutely ill, the fever is high and the pulse rate rapid. Abdominal tenderness and spasm are diffuse throughout; in some instances this spasm is of a "doughy" nature. The white blood cell count is often elevated between 20,000 and 40,000. The incidence of nephrosis in pneumococcal peritonitis is about 50 per cent.

The clinical picture differs somewhat in children with nephrosis; the onset is more insidious, and the abdominal pain is less severe and transient. A sudden rise in temperature accompanied by abdominal pain and vomiting in a child with nephrosis should suggest the diagnosis of pneumococcal peritonitis. Abdominal puncture is a useful, and often neglected, diagnostic aid. The purpose of abdominal puncture is to demonstrate the presence of intraperitoneal fluid in acute intra-abdominal lesions with signs of diffuse peritonitis. This permits de-

termination of the characteristics and bacteriology of the fluid. The technique is as follows:

> The puncture is usually made in the lower abdomen, lateral to the rectus muscle and on the side free of possible peritoneal adhesions, and well away from situation of a palpable or questionable mass. The skin of the abdominal wall is sterilized, and a small incision is made in the skin, after a wheal of local anesthesia, so as to obviate force in the introduction of the needle. A lumbar puncture needle with the stylet in place is used. As the resistance of the abdominal fascia and musculature is met and passed the needle enters the peritoneal cavity. The stylet is then removed and a syringe attached. Suction is used and continued as the needle is being withdrawn. Failure to obtain fluid does not exclude its presence. Often a drop or 2 is sufficient for study. Microscopic examination of the fluid is essential. It is not necessary to await a culture report, for a Gram stain of the smear will show the types of cells and microorganisms.

Frequent examination of the child who has sustained *nonpenetrating trauma* to the abdomen is important. Serious intra-abdominal injuries associated with nonpenetrating trauma are rupture of the jejunum, rupture of the duodenum, rupture of the spleen, and injury to the liver or mesentery. Signs of peritoneal irritation are common to all these injuries. Important aids to diagnosis in intestinal rupture are abdominal puncture (described above) and a plain roentgen film of the abdomen in the upright position which will demonstrate free air under the diaphragm. Splenic and hepatic injuries with intra-abdominal hemorrhage will produce progressive weakness, shock and anemia. The importance of careful observation is seen in the patient with delayed rupture of the spleen who suffers a subcapsular hemorrhage which ruptures after days.

Peptic ulcer, Meckel's diverticulum, regional enteritis, and *ulcerative colitis* often have complications—intestinal perforation, obstruction or hemorrhage—which require early surgical intervention. The previous history and radiologic findings will usually help to make the diagnosis. When the surgical complications occur, the signs of peritonitis, intestinal obstruction or hemorrhage are obvious.

Polyps of the colon (see also p. 1158) may produce abdominal pain associated with rectal bleeding. The diagnosis is made by radiologic examination of the colon. Multiple polyps of the small intestine and/or colon may produce the same symptoms, and in certain family groups are associated with dark brown spots on the lower lip and buccal mucosa of the mouth (*Peutz-Jeghers syndrome*).

Rarely, *acute pancreatitis* has been the cause of abdominal pain in childhood. This diagnosis is aided by the finding of an elevated amylase activity of the serum.

There are a number of *tumors and cysts of the intestinal tract and mesentery* producing a palpable abdominal mass, for which radiologic examinations of the intestine are necessary to make a diagnosis. *Ovarian*

cyst with twisted pedicle will be palpable on rectal examination, and visible by x-ray film.

Disorders of the retroperitoneal organs are frequent causes of abdominal pain in infancy and childhood. The abdominal symptoms of acute and chronic *urologic lesions* closely mimic abdominal disease. The genitourinary system should always be considered in the differential diagnosis of abdominal pain. A careful urinalysis is a necessary part of the examination of any child with abdominal pain. Related symptoms—nausea, vomiting and abdominal distention—are often present. Pain in the urinary tract is usually caused by *obstruction*. Among the most common abnormalities of the urinary tract producing abdominal pain are congenital obstructions at or near the ureteropelvic junction. These result in hydronephrosis, often with superimposed infection. Abdominal pain, nausea and vomiting are the most common complaints. Pyuria, chills or fever accompanies superimposed infection. Examination of the abdomen often reveals a mass. Excretory urograms are usually diagnostic, but occasionally must be supplemented by retrograde urograms. *Renal calculi* may produce abdominal pain by obstructing a calyx or the ureteropelvic junction. Hematuria or pyuria is frequent, and radiologic examination often shows the calculus. Nonopaque calculi can be diagnosed only by excretory or retrograde urograms. Other causes of abdominal pain are *pyelonephritis* and *renal trauma*. The pain of ureteral colic due to calculi is severe, intermittent, and usually accompanied by nausea and vomiting. If the calculus descends down the ureter, the site of pain varies from the costovertebral angle, then toward the anterior superior spine of the ileum, then the hypogastrium, and finally the leg or genitalia, accompanied by urinary frequency and urgency. Radiologic examination and excretory or retrograde urograms are diagnostic. Finally, obstructions of the bladder neck and urethra resulting in distention of the bladder may produce pain in the hypogastrium. In any child with a mass in the hypogastrium, catheterization of the bladder should be performed to be sure it is not a distended bladder.

In the period of early infancy acute surgical lesions are usually manifested by symptoms and signs of intestinal obstruction—*intussusception, intestinal atresias, malrotations, duplications* and *meconium ileus*. The differential diagnosis of these conditions depends upon the radiologic examination of the abdomen.

Abdominal tumors—Wilms's tumors, neuroblastomas and teratomas—are frequent in infants. In these tumors the abdominal mass is usually palpable. It is important that the radiologic examination include the chest, skull and long bones. The first clinical manifestation of these tumors may be metastases to one of these structures. We place these tumors in the category of urgent surgery.

Inguinal hernias are frequently associated with local pain, and when they are incarcerated, the pain is severe. (See also p. 970.)

PEDIATRIC DISEASES OF THE ABDOMEN

Acute gastroenteritis is a frequent, nonsurgical cause of abdominal pain in infancy and childhood. The abdominal pain and tenderness are usually diffuse and do not tend to localize. Acute gastroenteritis is generally accompanied by nausea, vomiting and diarrhea. The distinction from a surgical condition, such as acute appendicitis, depends upon careful observation. If nausea, vomiting and diarrhea persist, and the abdominal pain becomes less and does not localize, gastroenteritis should be considered the diagnosis and observation continued.

Gastrointestinal symptoms from *milk allergy* have been noted in infants. Colicky abdominal pain and diarrhea, the stool containing much mucus and sometimes blood, are characteristic of the intestinal allergy seen in young infants.

So-called *colic* in young infants is usually related to improper feeding and air-swallowing. However, a surgical lesion, such as intussusception, may produce this symptom, and must not be overlooked.

Anaphylactoid purpura (*Henoch's syndrome*) is characterized by abdominal pain associated with ecchymoses, petechiae and gastrointestinal hemorrhage (see also p. 1154).

A number of *parasitic diseases* may produce abdominal pain in children. The most common of these infestations are those of pinworms, roundworms, hookworms, trichinae, tapeworms and amebae. The diagnosis is made upon recovery of the characteristic worms or eggs in the stools.

A number of diseases produced jaundice (see also p. 1063) in infants and children—*bile-duct malformations* and *obstructions*, *cirrhosis* and *hepatitis*. These may be accompanied by abdominal pain, but the jaundice localizes the disease to the biliary system. The differential diagnosis depends upon laboratory examinations of the urine and stool, and liver function tests.

DISEASES REMOTE FROM THE ABDOMEN

There are diseases remote from the abdomen which may cause abdominal pain, and should be considered in children in whom the clinical picture is not clear-cut.

Pleuritic pain complicating pneumonia may be referred to the upper abdomen. Cough, rapid shallow breathing and grunting expirations should lead to a careful physical and radiologic examination of the

chest. The fever and white blood cell count are commonly high. The abdominal tenderness and spasm are usually subcostal.

Many *neurologic lesions* may cause abdominal pain. A tumor affecting one of the spinal nerve roots, Pott's disease of the spine, and herpes zoster affecting an abdominal nerve segment are examples. In these cases accurate localization of the lesion is usually possible, keeping in mind the distribution of the spinal nerves. Epileptic attacks have been manifested by abdominal pain. When this is part of a convulsive seizure, the diagnosis is clear, but in other cases the diagnosis rests upon the past history of seizures, and electroencephalographic abnormalities. Acute necrotizing diseases of the brain, such as bulbar poliomyelitis, may cause acute ulceration of the stomach or jejunum.

The child is known for his desire to swallow anything within reach, and a variety of *poisonings* are common. Lead poisoning is the most common heavy metal to produce abdominal pain. The diagnosis is established by examinations of the blood and urine concentrations of lead, and x-ray studies of the long bones.

Some of the so-called *collagen diseases*—rheumatic fever and periarteritis nodosa—may give rise to abdominal pain of no consistent type or pattern. These are generalized diseases of the connective tissues and small blood vessels of the body, and the major manifestations are found in other organs. The sedimentation rate is elevated in these conditions.

Abdominal pain may arise in children with *electrolyte disturbances* secondary to diarrhea or vomiting, producing acidosis, alkalosis or hypokalemia. In any child with vomiting or diarrhea, determinations of the electrolyte concentrations in the blood will determine the nature of the electrolyte disturbance, which can be corrected by adequate intravenous fluids. There may, of course, be an underlying condition which is the cause of the abnormality and needs treatment.

Diabetic acidosis is another metabolic disturbance causing abdominal pain, and a urinalysis for sugar and ketone bodies should be a routine part of the examination.

Porphyria is a hereditary disease of porphyrin metabolism in which there may be attacks of abdominal pain. During attacks the urine may be red. Even when the urine has a normal appearance it contains, during the paroxysms of abdominal pain, large amounts of porphobilinogen, a colorless metabolite. When porphyria is suspected, the diagnosis should be confirmed by expert analysis of the urine.

Abdominal pain may be a symptom in a number of *hematologic diseases*. Examples are leukemia, hemolytic anemias, transfusion reactions, polycythemia vera and hemophilia. In some instances the pain may be due to splenic infarcts or hemorrhage into the bowel wall; in others to a blood destruction. Sickle cell anemia is an inherited ab-

normality of hemoglobin limited almost exclusively to Negroes, characterized by sickle-shaped red blood cells. During a "crisis" the picture may be that of an acute "surgical abdomen." In all these conditions an accurate blood examination is diagnostic.

There are children with recurrent attacks of abdominal pain for which, after thorough investigation, no cause can be found. Some of them may have serious *emotional problems* caused by anxiety, fear, resentment and frustration, or the malingerer who wishes to escape responsibilities. Such a child needs psychiatric help.

SUMMARY

An analysis of abdominal pain in children and the details of abdominal examination are presented. A method of clinical analysis of children with this complaint is reviewed by a discussion of the many surgical and pediatric diseases of the abdomen and the remainder of the body which produce abdominal pain.

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EDITOR'S NOTE. A discussion of abdominal pain in children is a tremendous subject to encompass in a few pages. Obviously, an effort such as this cannot take into consideration the multitudinous variations presented by some of the medical conditions of youngsters producing abdominal pain.

Abdominal pain is certainly one of the most common symptoms in childhood. Indeed, children probably have abdominal pain as often as adults feel malaise. Therefore the diagnostician must not only be aware of the fact that abdominal pain can be a prodromal symptom of practically any pediatric disease, but must also be aware of the fact that the same symptom can usher in a surgical situation requiring prompt action. To complicate further the diagnosis of medical and surgical abdominal pain, it must be remembered constantly that it is possible to have acute appendicitis in the presence of any other medical pediatric problem. Indeed, many of the infectious diseases in children, producing edema and hyperplasia of mesenteric lymph nodes as well as of the appendiceal lymphoid follicles, can produce a type of appendicitis which is pathologically a true appendiceal folliculitis.

Increasing experience with the vagaries of abdominal pain in children certainly improves one's diagnostic ability, and with the passage of the years the number of laparotomies undertaken for nonsurgical disease should certainly decrease.

The use of needle puncture in the diagnosis of peritonitis is certainly of great benefit in a limited number of patients, as has been indicated, but also carries with it the danger of perforation of the bowel. It has been extremely helpful in certain categories of patients, particularly in postoperative neurosurgical patients in whom the differential diagnosis between postneurosurgical perforation of the bowel and appendicitis is in question.—C. E. K.

LESIONS ABOUT THE UMBILICUS IN INFANTS AND CHILDREN

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After the initial care has been given to the umbilical cord of the newborn infant the umbilical region usually receives little further consideration. There are, however, many congenital and acquired lesions about the umbilicus with which physicians must be concerned. Certain of these, e.g., granuloma of the umbilicus and umbilical hernia, are easily diagnosed and treated, but even with these more common lesions there is little unanimity of opinion about the proper method of therapy. The rare umbilical lesions such as omphaloceles and remnants of the urachal or omphalomesenteric duct are no less important. They are encountered by the obstetrician, the pediatrician, the general practitioner and the general surgeon. Immediate recognition and treatment of some of these lesions are of paramount importance to the welfare of the child.

There have been many publications concerning specific lesions in the umbilical region since the complete treatise of Cullen[24] was published in 1916. It is hoped that the classification of umbilical lesions in children (Table 1) will be of value in the interpretation of lesions in this area. Since a clear understanding of the embryologic development of the umbilical area is essential to an understanding of its pathologic conditions, a review of the embryology is included.

EMBRYOLOGY

The anatomy and anomalies of the umbilical region are best understood if the development of the embryo is followed through the tenth week

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TABLE 1. *Classification of Lesions in the Region of the Umbilicus*

I. Alimentary anomalies
A. Completely patent omphalomesenteric duct (umbilical enteric fistula)
B. Partially patent omphalomesenteric duct
1. Peripheral portion (umbilical sinus)
2. Intermediate portion (vitelline cyst)
3. Enteric portion (Meckel's diverticulum)
C. Congenital band (obliterated omphalomesenteric duct)
D. Mucosal remnant at the umbilicus (umbilical polyp)
II. Urachal anomalies
A. Completely patent urachus (umbilical urinary fistula)
B. Partially patent urachus (urachal cysts)
1. Peripheral portion (umbilical urachal sinus)
2. Intermediate portion (urachal cyst)
3. Vesical portion communicating with the bladder
III. Vascular anomalies
A. Persistent omphalomesenteric vessels
B. Persistent umbilical vessels
C. Persistent urachal vessels
IV. Somatic anomalies
A. Umbilical hernia
B. Omphalocele
C. Exstrophy of the bladder
D. Exstrophy of the cloaca
E. Gastroschisis
F. Epigastric hernia
G. Diastasis recti
V. Acquired lesions of the umbilicus
A. Infections
1. Acute
2. Chronic
B. Granuloma
C. Hemorrhagic lesions
D. Traumatic lesions
E. Concretions
VI. Tumors of the umbilical area

Modified from Trimingham and MacDonald.[98]

of intrauterine life. The reader is referred to Arey,[4] Begg,[7, 8] Bremer,[13] Cullen[24] and Trimingham et al.[98] for a more detailed study of the embryology of the umbilical region.

Formation of the Umbilical Cord

The 3-week embryo has a dorsal cap of amnion and a relatively large yolk sac on its ventral surface.[24, 98] The embryo develops from the embryonic plate to which both of these structures are attached. As the embryo starts to enlarge, the amnionic sac encircling the embryo from its ventral surface indents the base of the yolk sac at its attachment to the embryo. The celomic cavity which is formed in this manner consists of the residual space between the base of the yolk sac and the ventral wall of the embryo. As the embryo and the amnionic sac continue to grow, the yolk sac is forced away. The narrow portion of the yolk sac is now called the omphalomesenteric or vitelline duct. The center of the yolk sac and the lumen of the digestive tract are connected through this duct.[4] The foregut and the hind gut develop from that portion of the yolk sac which is drawn into the head fold and the hind fold of the embryo.[13] The

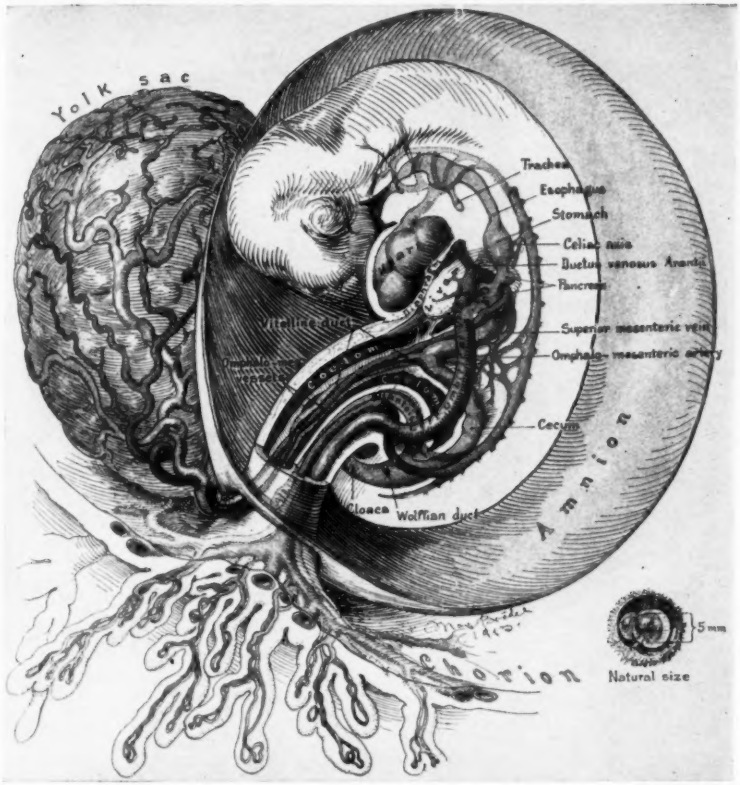

Fig. 49. Sagittal section of human embryo 4 weeks of age and the umbilical cord. (Cullen.)

digestive tract ends in the hind gut as the cloaca. From the cloaca the allantois projects into the body stalk, which consists of the point of attachment of the embryo to the placenta and contains the umbilical vessels. The body stalk becomes elongated as the embryo develops. The foregut divides into 2 parallel tubes.[13] The more ventral of these forms the trachea, and the dorsal half of the foregut forms the esophagus and upper intestinal tract. The cloaca is developed from the hind gut and is divided into the dorsal rectum and the ventral urogenital sinus. This division is produced from above downward by the paired lateral folds of the mesoderm terminating at the cloacal plate.

An umbilical cord is produced early in the fifth week[4, 24] (Fig. 49) as the omphalomesenteric duct and the body stalk are pressed close together and elongated by that portion of the amnionic sac which encircles and covers these structures.

Urachus

The urachus, it is thought by some,[13, 24, 41, 97] is derived from the superior part of the allantois. Others,[4, 7, 8] however, believe that the urachus and bladder develop solely from the ventral cloaca. The bladder and urachus elongate with the progressive development of the infraumbilical abdominal wall.

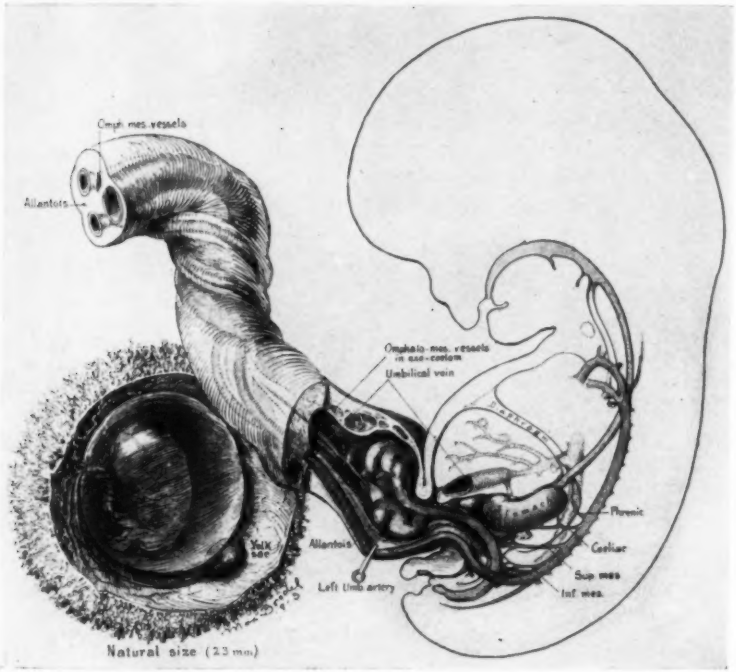

Fig. 50. Sagittal section of the umbilical cord of human embryo 8 weeks of age. (Cullen.)

The urachus is located between the peritoneum and the transversalis fascia and extends for a distance of about 1 cm. into the umbilical cord along with the umbilical arteries and vein. In the cord the urachus is attached to the obliterated end of the allantois. At birth the length of the urachus is only 2 to 2.5 cm. from the bladder to its termination in the umbilical cord.[6, 8]

At birth the termination of the umbilical arteries and the urachus at the umbilicus becomes involved in the extensive fibrous tissue which closes the umbilical fascial tunnel and forms the umbilical scar.[8] In the newborn the true pelvis is underdeveloped; little room is available for the bladder. After birth the bladder descends into the pelvis. During the descent of the bladder the urachus is pulled down. The obliterated umbilical arteries are pulled caudad as the urachus and bladder descend into the pelvis. They become anchored to the anterior abdominal wall several centimeters above the apex of the bladder and help to control its emptying. The urachus persists throughout life as a cord, the inferior portion of which is patent in about 33 per cent[8] of persons and extends one third of the distance from the bladder to the umbilicus.[4]

Omphalomesenteric Duct

As the digestive tract continues to grow, the size of the celomic cavity does not keep pace with the enlarging liver. As a result, the intestinal loops are forced out into the umbilical extraembryonic celomic cavity. They are accompanied by the omphalomesenteric vessels, which are derived from the yolk sac, and ultimately these vessels communicate with the superior mesenteric vessels. The intestines coil (Fig. 50) in this

rapidly enlarging space. The colon slowly increases in length and begins a counterclockwise rotation. The cecum thus rotates to the right side of the coils of the small bowel. At the end of the tenth week,[4, 13] as a result of the relative decrease in size of the liver and the rapid expansion of the abdominal wall, the intestines return to the abdominal cavity. The extracelomic cavity in the umbilical cord is then obliterated by encroachment of the mesodermal tissue of the cord.[4]

ALIMENTARY ANOMALIES

COMPLETELY PATENT OMPHALOMESENTERIC DUCT

The omphalomesenteric duct usually disappears during the fourth to seventh week of embryonic life.[24] If obliteration fails to occur, various anomalies may persist. A completely patent omphalomesenteric duct results from total failure of the duct to close. The resultant patent duct becomes apparent when the umbilical cord separates, and a fecal discharge is usually noted at the site of the umbilicus. The patent omphalomesenteric duct is far more frequent in the male than in the female,[24, 91] the ratio being nine males to one female.[73] The diagnosis is usually made during the first year of life and is usually manifest in the neonatal period[91] (Figs. 51, 52).

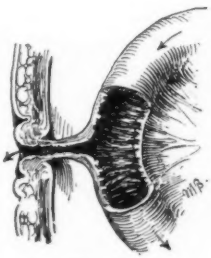

Fig. 51.

Fig. 52.

Fig. 51. Completely patent omphalomesenteric duct. (Cullen.)

Fig. 52. A gross specimen of a completely patent omphalomesenteric duct from a stillborn.

Symptoms and Diagnosis

When a patent omphalomesenteric duct is present, the base of the umbilical cord is larger than normal. The umbilical depression is usu-

ally occupied by a bright red nodule which varies considerably in size. Fecal discharge from the fistula is the most common finding, the size of the duct governing the volume and frequency of the drainage. The skin around the fecal fistula is usually irritated. If the patent duct is untreated, prolapse of the small bowel may occur through the duct during infancy.[24] The diagnosis of this anomaly is made by injecting radiopaque material into the duct and visualizing its course and communications with the bowel on lateral roentgenograms of the abdomen.

Treatment

The parents of infants with a persistent omphalomesenteric duct should be warned that intestinal obstruction from the bowel twisting around the intra-abdominal portion of the duct or from prolapse of the bowel through the duct may occur if surgical correction is postponed or declined.[51] The best results are obtained by excision of the umbilicus and removal of the fistulous tract. The intestinal portion of the tract should be inverted. Since a prolapse in this area is usually impossible to reduce, operation should be performed as soon as the diagnosis is established and before prolapse can take place.[21, 30, 50, 91]

PARTIALLY PATENT OMPHALOMESENTERIC DUCT

Peripheral Portion—Umbilical Sinus. This condition exists when the omphalomesentric duct becomes obliterated intra-abdominally, but remains patent distally. The sinus tract thus communicates with the umbilicus, but not with the intestines (Fig. 53).

A polypoid mass is first noted at the umbilicus when the umbilical cord separates and a discharge of mucoid material exudes from the umbilicus. This umbilical mass varies from one to several centimeters in diameter and contains a central canal.[24] A probe can usually be passed 1 to 2.5 cm. into this canal. The mucosa of the polypoid mass resembles that of the small bowel.

The *diagnosis* may be suspected by the mucoid characteristic of the discharge. It can be confirmed by injection of the tract with radiopaque

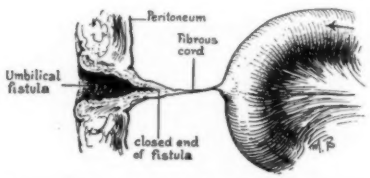

Fig. 53. Partially patent omphalomesenteric duct illustrating persistence of the peripheral portion of the duct. (Cullen.)

material and visualizing the tract on lateral roentgenograms of the abdomen.

Treatment is by excision of the umbilicus and the patent duct.

Intermediate Portion—Vitelline Cyst. Vitelline cysts occur when the intermediate portion of the omphalomesenteric duct fails to obliterate between the small bowel and the umbilicus (Fig. 54). These cysts are lined by intestinal mucosa. The cyst may enlarge as the result of accumulation of secretion, and ultimately the epithelium may be obliterated as a consequence of increased intraluminal pressure.

Since a fibrous connection persists between the umbilicus and the intestine, these cysts are associated with the same dangers of intestinal obstruction as are present with congenital bands (see below). The cyst alone usually causes no symptoms, but larger ones may be detected by palpation or inspection.[89] The cyst should be excised.

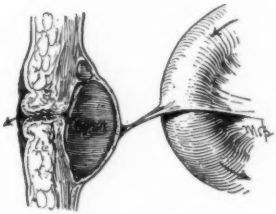

Fig. 54. Small cyst of the umbilicus due to patency of the intermediate portion of the omphalomesenteric duct. Note the persistence of the obliterated omphalomesenteric duct. (Cullen.)

Enteric Portion—Meckel's Diverticulum. A Meckel's diverticulum is the result of persistence of the intra-abdominal portion of the omphalomesenteric duct. The diverticulum usually projects from the antimesenteric border of the ileum, but has been found in the jejunum as well. The lining may contain pancreatic tissue, gastric, small intestinal or colonic mucosa.[101] The diverticulum may be long or short and may be attached to the umbilicus or mesentery by a fibrous band which represents obliterated omphalomesenteric vessels (Fig. 55). In some instances the diverticulum has its own mesentery.[24] (See also p. 1156.)

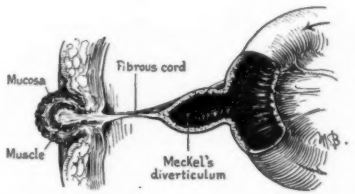

Fig. 55. Enteric portion (Meckel's diverticulum). Note umbilical polyp which may be connected to Meckel's diverticulum by a fibrous band. (Cullen.)

CONGENITAL BAND—OBLITERATED OMPHALOMESENTERIC DUCT

Congenital bands may exist as a remnant of an obliterated omphalomesenteric duct or of the omphalomesenteric vessels. These bands connect the umbilicus with the antimesenteric border of the small bowel. As a result, a loop of bowel may become twisted around the fibrous cord and cause intestinal obstruction.[24, 73] These bands should be surgically excised. Caution should be exercised during this procedure, since the intestinal end of the band may communicate with the lumen of the intestine.

MUCOSAL REMNANT AT THE UMBILICUS (UMBILICAL POLYP)

The remnant of the mucosa of the omphalomesenteric duct may appear as an umbilical polyp or mass. The polyp or mass is covered by intestinal mucosa and secretes mucus (Fig. 56). These remnants make their appearance after the cord has fallen off. The secretion of mucus differentiates the umbilical polyp from granulation tissue, and the absence of an opening excludes a patent omphalomesenteric duct.[24, 51]

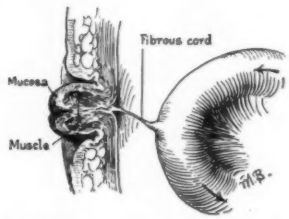

Fig. 56. Umbilical polyp which may be connected to the small bowel by a fibrous cord. (Cullen.)

Features in the differential diagnosis of granulation tissue from umbilical polyp[51] are as follows:

GRANULATION TISSUE	UMBILICAL POLYP
1. Only in first few weeks of life	1. May persist for years
2. Dull red or pink	2. Bright red
3. Soft	3. Firm and resistant
4. Purulent secretion	4. Usually a mucoid secretion
5. Histologically—granulation tissue	5. Histologically—intestinal tissue
6. Can be successfully treated by local application of astringents	6. Unaffected by astringents—should be removed by surgical excision

URACHAL ANOMALIES

Anomalies of the urachus develop because of failure in its descent or formation. Four types of urachal anomalies may be encountered as

indicated in Table 1. In the anomalies of descent the urachus may remain patent throughout its length with a resultant umbilical urinary fistula. A partially patent urachus may persist as a peripheral or umbilical urachal sinus. If the intermediate portion of the urachus remains patent, a closed urachal cyst develops. When the communication between the bladder and the urachus persists, a vesical urachal cyst is present.

COMPLETELY PATENT URACHUS—UMBILICAL URINARY FISTULA

A patent urachus is manifest a few days after the cord has separated, at which time persistent drainage of urine from the umbilicus is noted. This drainage may be intermittent or continuous and varies from small to larger amounts. The characteristics of the drainage should differentiate a patent urachus from a persistent omphalomesenteric duct or from a granuloma of the umbilicus.

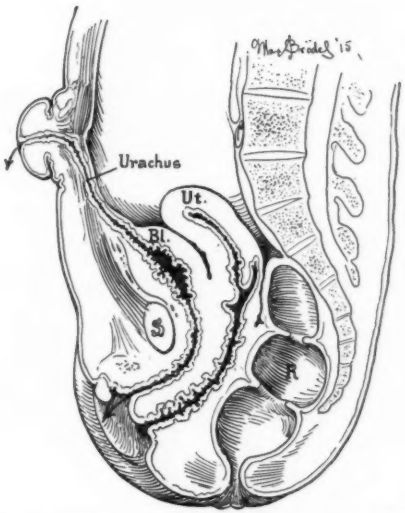

Fig. 57. Completely patent urachus in a female. (Cullen.)

Patients with a patent urachus may have little alteration of their umbilicus, or a small mass may persist at the umbilicus. The umbilical abnormality may present as a small globular mass, or there may be a protrusion that resembles a penis or cock's comb.[7, 24] This umbilical mass, which is the prolapsed distal portion of the urachal duct, is covered in part by urachal mucosa and in part by skin (Fig. 57).

The opening of the sinus is usually situated in the most prominent

point, i.e., at the center of the umbilicus or protrusion. Its size varies from a small pinpoint orifice to an opening through which a catheter can be passed readily. Occasionally, in spite of a large opening, a catheter cannot be passed through the tract because of its tortuosity.

The skin surrounding the opening of the sinus usually is irritated, but this depends on the amount of urine flowing from the duct. In general, however, the degree of irritation is not as great as it is in a fecal fistula. If infection is present, the appearance of the skin of the umbilicus and characteristics of the drainage fluid will vary with the severity of the infection.[77]

The *diagnosis* can be made by injection of radiopaque material into the sinus followed by lateral roentgenographic studies of the abdomen. This will establish the presence of a connection with the bladder. A cystogram as well as a voiding urethrogram should be performed to establish the diagnosis in a retrograde manner.[35] In some instances the patent urachus will be filled in a retrograde manner by these techniques, which will also be of value in demonstrating the presence or absence of obstruction at the urethra or bladder neck.

Incidence

This rare anomaly has been found 23 times in 15,919 autopsies in children.[17] Nix et al.[77] report a sex incidence of nine males to one female.

Treatment

Many methods of treatment of patent urachus have been reported. Extraperitoneal excision, however, appears to be the treatment of choice. Surgery should be carried out as soon as the diagnosis is made, since delay may be associated with infection and an increased risk to the patient. Moreover, if it is not excised, a small number of patients with a persistent urachus have subsequently had carcinoma at this site in later adult life.[1, 80, 84, 90, 93, 105]

Excision of the umbilicus and the urachal tract to the apex of the bladder is performed through a midline incision. An indwelling catheter in the duct will facilitate excision. If the duct is small, injection with methylene blue will assist in its identification. The stump at the dome of the bladder should be excised and the bladder closed with interrupted sutures.

PARTIALLY PATENT URACHUS (URACHAL CYST)

Urachal cysts are not diagnosed unless they are large or become infected.[15, 32] These cysts are remnants of the urachus in its descent[8, 24]

and may occur at any age, but are most common in children.[57] The fluid content of a urachal cyst may be urine, mucoid fluid from the wall of the cyst, serous transudate, pus, blood, cholesterol, fat cells, epithelium or fibrin.[32] A blind cyst develops when both ends of the urachus close and the intermediate portion of the duct develops into a cyst. Communicating cysts may connect externally with the umbilicus or internally with the bladder.

Symptoms

When the cyst attains a large size, there is a palpable midline mass. The cyst lies between the peritoneum and the abdominal muscles and may cause abdominal pain as a result of pressure, or urinary symptoms may be manifest if it becomes infected. Malek and Boulgakow[68] reported a huge cyst 6 by 10 cm. associated with absence of the urethra in a 6-months' fetus. Urachal cysts may rupture into the umbilicus, bladder or peritoneal cavity. In one of our patients the infected cyst ruptured into both the umbilicus and the peritoneal cavity (Fig. 58). At

Fig. 58. Lateral roentgenogram of the abdomen after injection of an umbilical sinus with Lipiodol. A fine tract can be seen extending from the umbilicus and ending in a ruptured cyst that perforated into the peritoneal cavity.

operation, after injection of Lipiodol into the umbilical opening, a sinus tract was found extending from the umbilicus to a cyst which had ruptured into the peritoneal cavity. No previous umbilical discharge had been noted in this patient.

Infected cysts are found more commonly because they give rather pronounced symptoms, such as pain in the lower abdomen, often more pronounced with voiding, fever, generalized malaise, gastrointestinal upsets and frequency of urination.[32] There is usually a rather sudden

onset of these symptoms. The finding of a cystic mass between the umbilicus and the symphysis pubis suggests a noninfected urachal cyst. An infected urachal cyst, in addition, is tender to palpation, and there is associated inflammation of the anterior abdominal wall.

Incidence

Urachal cysts are more common in the male;[48] Campbell[17] found that they occurred in three males to one female. This lesion was found in only 3 of 15,919 autopsies in children.[17]

Differential Diagnosis

The differential diagnosis includes such lesions as appendicitis, Meckel's diverticulitis, distended bladder and ovarian cyst.[38]

Catheterization of the bladder should be performed in an attempt to establish the nature of any midline mass. If this mass disappears after catheterization and subsequently recurs, then cystoscopy and a cystogram may be helpful in demonstrating a urachal cyst which drains into the bladder.[35] If the cyst drains via the umbilicus, lateral films of the abdomen after injection of the sinus will establish the diagnosis (Fig. 59). If infection is present, the differential diagnosis may be difficult.

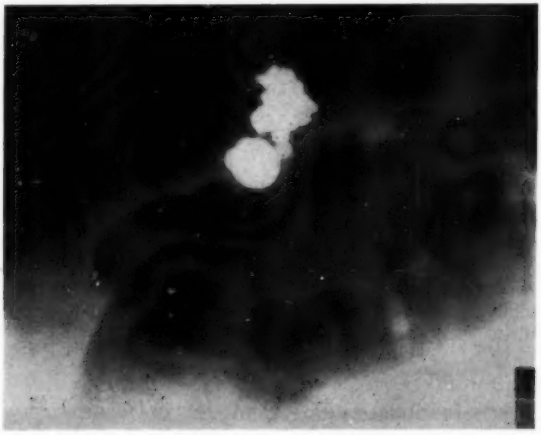

Fig. 59. Lateral roentgenogram of the abdomen taken after injection of the umbilical sinus with Lipiodol. The sinus connects with an underlying cyst.

Treatment

Primary excision is the treatment of choice for uninfected urachal cysts. An infected urachal cyst, however, should be incised and drained.

After infection has subsided a secondary operation with excision of the cyst wall is advocated.[38, 45, 60, 83, 88] Excision of the cyst should be carried out after incision and drainage even if there is no evidence of a residual cyst, since the incidence of carcinoma derived from these urachal remnants is an appreciable hazard in later life.[1, 80, 84, 90]

VASCULAR ANOMALIES

The two umbilical arteries of the embryo are located on either side of the urachus and empty into the anterior division of the internal iliac arteries. After birth they atrophy and form the cordlike structures known as the obliterated hypogastric arteries. The right umbilical vein disappears before the embryo reaches 10 cm. in length. The left umbilical vein persists as the umbilical vein of the fetus, draining directly into the liver; after birth it becomes the ligamentum teres. Structural abnormalities of these vessels are extremely rare. Their main importance is that they may serve as avenues for the spread of infection. Omphalitis and its sequelae are discussed elsewhere (p. 1109).

The omphalomesenteric vessels which supply the circulation for the yolk sac and the omphalomesenteric duct in fetal life normally disappear before birth. Their remnants, however, will occasionally form a fibrous band running from the umbilicus to Meckel's diverticulum and will thus serve as a point around which loops of intestine may become caught. The omphalomesenteric artery may remain patent when it accompanies a persistent omphalomesenteric duct. The spurt of blood which occasionally is seen when an umbilical polyp is removed is said to come from a persistent omphalomesenteric artery.[24]

Persistence of the urachal vessels may occur when a patent urachus exists and produces a granuloma of the umbilicus.[8]

SOMATIC ANOMALIES

UMBILICAL HERNIA

Definition

As used in this paper, an umbilical hernia is an incomplete closure of the fascia of the umbilical ring, through which protrusion of intra-abdominal contents may occur. The area is covered by skin, a condition which differentiates an umbilical hernia from an omphalocele.

Etiology

No common etiologic factor is known to account for the occurrence of umbilical hernia in the majority of patients, but certain specific conditions are associated with the presence of umbilical defects.

Premature infants have an increased incidence of umbilical hernia, ranging from 84 per cent in newborn infants weighing 1000 to 1500 gm., to 20.5 per cent in infants weighing 2000 to 2500 gm.[33] By contrast, however, Woods[104] noted an increased incidence of umbilical hernia in infants who weigh over 3200 gm. at birth. He postulates that the persistent defect may be due to the large size of the umbilical cord. He and others note that there is a familial tendency to the occurrence of the umbilical hernias.[53, 58]

Some specific diseases with which umbilical hernias are frequently associated include mongolism, gargoylism, amaurotic familial idiocy[53] and cretinism. Specific thyroid therapy for the latter may result in disappearance of the hernia.[85]

Any condition which increases intra-abdominal pressure may cause an umbilical hernia, e.g., ascites from any cause, chronic constipation or coughing,[22, 104] the celiac syndrome or pregnancy. Sepsis of the umbilical stump is also cited by Woods[104] as being an etiologic factor. Crump[22] states that among certain tribes along the Gold Coast of Africa, traction is used on the umbilical stump in infants in order to produce large umbilical hernias, which are considered a mark of beauty.

Incidence

The incidence of umbilical hernia in normal full-term infants varies with the race and age of the patient. Several excellent studies[22, 33, 53, 55, 56, 58, 104] indicate a definitely higher incidence in the Negro race and a lower incidence in the Chinese and Indian races.[56] Spontaneous regression of the umbilical hernia occurs normally in the majority of patients, being most rapid during the first year of life.

Unfortunately no one thus far has followed up a significant number of patients from birth through parenthood to determine accurately the incidence or significance of umbilical hernias; each of the previously mentioned studies was performed on different groups of patients at various age levels. Studies by Crump[22] in this country indicate that the incidence of umbilical hernia in Negro infants under 1 year of age is 41.6 per cent and that this decreases steadily to 15.9 per cent at 4 years of age and essentially zero after 8 years of age. It is doubtful, however, whether the number of patients studied after 2 years of age is of statistical significance. Evans[33] studied 1339 Negro infants under a year of age and found an incidence of umbilical hernia of 31.8 per cent in patients up to 6 weeks of age; this decreased to 9.5 per cent by 26 weeks of age and rose again to 12.6 per cent at 1 year of age. These figures contrast with an incidence of 4.1 per cent in full-term white infants, followed by a fall to 1.9 per cent by 1 year of age. The number of patients studied in each period after 6 weeks of age, especially in the series of white children, is not large enough to be statistically significant. In both Evans'[33] and Crump's[22] series there is an equal incidence in males and females.

Indications for Treatment

The lack of knowledge of the natural history of umbilical hernia contributes to the confusion as to the proper therapeutic approach. Only four reports of strangulated umbilical hernia in infancy could be found in the English literature,[44] but incarceration occurs somewhat more frequently. Most of the members of the Section on Surgery of the American Academy of Pediatrics report several instances in their experience. On the Surgical Service of St. Christopher's Hospital for Children we have encountered four patients with incarcerated umbilical hernias and one in whom omentum had strangulated into the hernia after a

burn and ulceration of the umbilical hernia sac. A history suggestive of intermittent incarceration was obtained in several other older children with small defects in the umbilical fascia. Spontaneous rupture of umbilical hernia in infancy has been reported twice.[67, 95] In each instance the infant was three months of age and had no other known abnormality. Other conditions which necessitate early surgical correction are rapid enlargement, the presence of a thin skin overlying the sac in an active infant (Fig. 60), ulceration of the skin and irritation from trauma; the last is often self-inflicted by an inquisitive child. The coincidental repair of an umbilical hernia during an operation for inguinal hernia is practiced by some surgeons.

Fig. 60. An umbilical hernia in a 7-month-old female Negro. Note the thin skin covering the hernia. Surgical correction was advised because the infant irritated the overlying skin by pulling at it.

Treatment

There is no consensus as to the best method of elective therapy for umbilical hernia. Adhesive strapping is recommended by as many authors as it is condemned as useless or even harmful by others. There is some agreement that there is no beneficial effect from strapping after the infant is six months of age. Woods[104] followed up 122 patients with umbilical hernia and found that 93 per cent of the defects closed spontaneously. He believes that strapping may delay spontaneous closure. Haworth[44] alternately strapped the umbilical hernia of 100

infants and reports that 80 per cent of the defects had closed by one year of age in the 50 infants who were strapped, but in only 43 per cent of those in whom they were not strapped did the defect close spontaneously. In accord with Cucinotta's[23] and our own experience at Temple University Hospital and St. Christopher's Hospital for Children, we believe that strapping rarely hastens the natural progress to closure of an umbilical hernia. It has, however, definite value in pacifying anxious parents and physicians. If there is any merit to strapping, it should be properly applied by using a tongue-and-groove technique as described by Gross[41] and Swenson.[97] At least, adequately long strips of waterproof adhesive should be used, and the skin over the rectus muscles should be approximated with the umbilical hernia inverted between them. Tincture of benzoin should be applied to the skin to protect it from irritation. The use of transparent cellophane tape as described by Prince[81] would seem to have the advantage of cleanliness; moreover, the transparency of the tape enables one to evaluate the degree of irritation of the skin beneath the dressing.

The ideal time to repair a persistent umbilical hernia electively is unknown. Although complications are rare, and a number of the hernias will regress during early childhood, we have no accurate knowledge of the number of these which may reappear in adult life and cause serious difficulties. A questionaire was sent to the members of the Section on Surgery of the American Academy of Pediatrics about this problem. Most of the 50 members who replied stated that their indications as to the age for elective surgical repair were becoming more conservative. Only four surgeons recommended elective repair under one year of age. Seven preferred to wait until the child was between three and five years of age. The majority of this group, including ourselves, adopt a flexible policy and attempt to individualize the condition. If a defect 1.5 to 2 cm. in diameter at one year of age has not been regressing, elective repair is recommended. A persistent defect of 1.5 cm. at 2 years of age, especially if there is protrusion, is repaired, and any umbilical hernia in a child over 3 years of age should be repaired. Many surgeons are more inclined to recommend surgical repair of an umbilical hernia in females because of the increased possibility of its reappearance in later life during pregnancy. (See also p. 996.)

The surgical technique used at St. Christopher's Hospital for Children for the correction of umbilical hernia is modified from that described by Blodgett,[10] Gross[41] and Swenson.[97] A semicircular incision is made in the redundant skin of the umbilicus, usually inferior to the fascial defect, and the umbilicus is preserved. Large sacs are removed, and the freshened edges of the posterior rectus sheath and peritoneum are approximated vertically as a single layer. The edges of the anterior rectus sheath overlying the medial border of the underlying rectus muscles are approximated vertically, and any associated diastasis recti is thereby corrected. Stainless steel wire sutures size no. 32 or 35 are preferred.

The Mayo technique for the repair of umbilical hernia is equally

popular with members of the Section on Surgery of the American Academy of Pediatrics. This method must be used for huge defects such as those seen in gargoylism or chronic recurrent ascites, but the associated diastasis recti cannot be corrected when this technique is used. Christopher[19] stresses the importance of placing interrupted sutures between the mattress sutures which are used in order to anchor the inferior flap of the fascia to the posterior surface of the superior flap.

OMPHALOCELE

Definition

The term "omphalocele" refers to a congenital defect of the abdominal wall through which intestines and other intra-abdominal organs protrude outside the abdominal cavity. The viscera are covered by the thin transparent amnion.[63, 82, 98] The absence of skin and peritoneum differentiates this condition from the common umbilical hernia. A variety of other terms have been used to identify this lesion in the English literature. These include exomphalos, congenital umbilical hernia, amniotic hernia, amniocele, funicular hernia of the umbilicus, ectopia viscerum, umbilical eventration, and hernia into the umbilical cord. This condition does not represent a true hernia because, on an embryologic basis, the displaced viscera have never returned into the abdominal cavity, and there is no true peritoneal sac about them.

Incidence

The variety of synonyms for this condition makes it difficult to evaluate accurately its incidence. Jarcho[54] reviewed several series of reported cases and found an incidence of 1:6600 births. If this incidence is correct, more than 600 infants with omphaloceles are born each year in the United States, an incidence equivalent to that estimated for cystic fibrosis of the pancreas.

The condition is not usually hereditary.[54, 86] Rothenberg,[86] however, stated that the patient which he reported was the third reported incidence of the occurrence of this anomaly in siblings. Males were affected in the proportion of two to one in O'Leary's collected series[79] and in the authors' series of seven cases. Most authors, however, have found the sex incidence to be equal.[63, 66] The incidence of prematurity in patients with omphalocele is no higher than the expected incidence of prematurity.[63]

Clinical Findings

The presence of an omphalocele in a fetus has been diagnosed by Fawcitt[34] from examination of an abdominal x-ray film of the mother.

Fig. 61. Newborn with a large omphalocele. Fascial defect measured 8 cm. in diameter, and the membranes are intact.

Fig. 62. Lateral roentgenogram of a newborn male who had intestinal obstruction shortly after birth. This film shows a loop of bowel caught in a small unrecognized omphalocele which had been inadvertently tied off.

Its presence rarely interferes with the infant's delivery,[20] although Warman[99] and Hemming[46] did experience difficulty in the delivery of affected infants.

The anomaly is obvious at birth (Fig. 61). The infant has a defect in the abdominal wall at and above the umbilicus through which various abdominal organs protrude. They are covered only by a thin, transparent membrane of amnion. Omphaloceles constitute one of the few true surgical emergencies of the newborn period. As the infant cries

and swallows air the intestines become inflated and further distend the sac. Within a few hours the membrane becomes dry and opaque, and cracks form in it through which bacteria invade the underlying peritoneal cavity.

In approximately 20 per cent of the cases the protective sac will be ruptured at time of delivery.[41, 66] If the rupture has occurred before birth, the viscera are matted together by firm leathery adhesions. When rupture of the membrane occurs during delivery, the intestines rapidly fill with air and spill out of the abdomen with secondary contamination of the peritoneal cavity.

Fig. 63. Roentgenogram of a newborn with an omphalocele, seen as the circular area of increased density in the center of the film. Note gas-filled loops of bowel above the diaphragm on the left side. This represents a diaphragmatic hernia through the foramen of Morgangi.

The size of the defect varies from a few centimeters in diameter to a total absence of the abdominal wall, the average size being 3 to 8 cm. in diameter. The smaller defects, which are usually limited to the umbilicus, are commonly designated as umbilical cord hernias. Their presence may be unrecognized when the cord is tied,[79] and bowel obstruction with the formation of a fecal fistula may follow (Fig. 62).

Associated Anomalies

Associated congenital anomalies are present in 59 per cent of patients with omphaloceles.[5, 41] Many of these anomalies are not of immediate

significance, i.e., harelip, cleft palate, inguinal hernia, undescended testes, club feet, and so on. Recognition of the more severe associated congenital anomalies, however, is important, since they may modify the surgical procedure. The authors have encountered one infant with an omphalocele who also had a diaphragmatic hernia which was successfully repaired at the time of closure of the abdominal wall (Fig. 63). McKeown[66] reported 4 patients with omphalocele and diaphragmatic hernia in a series of 69 cases. Other severe anomalies which have been present in patients with omphalocele include stenosis or atresia of the intestine,[9, 12, 14, 36, 61, 92] persistent omphalomesenteric duct,[2, 9, 39, 71] Meckel's diverticulum,[9, 74] duplication of the intestine,[3] volvulus of the midgut,[41, 47] malrotation of the cecum and obstructing bands,[27, 28, 41] hydronephrosis,[62] exstrophy of the bladder,[59] congenital heart disease,[41] meningocele,[41] anencephaly[87] and others.

Treatment

The commonly accepted method of treatment of omphalocele is immediate surgical repair of the defect. The earlier the repair is accomplished, the less time there is for the intestine to fill with air and for drying and for contamination of the thin avascular amniotic sac. A review of the many isolated reports of the successful repair of omphaloceles emphasizes the fact that all physicians who practice obstetrics, pediatrics or surgery must be prepared to advise corrective surgery immediately after the infant has been born. An attempt should be made to repair all these defects if there is no obvious associated congenital anomaly incompatible with life.

Preoperative treatment consists in covering the sac with warm sterile saline- or Zephiran-soaked gauze pads to prevent contamination of the peritoneum. A nasogastric tube is inserted into the stomach and placed on continuous suction to diminish the amount of air passing into the intestines. Chemotherapy and vitamin K are administered intramuscularly, and a plastic tube is placed in one of the infant's veins for administration of blood and fluids. A careful evaluation of the patient for the presence of other congenital anomalies is imperative. Because a diaphragmatic hernia or atresia of the intestines may be present in association with the omphalocele, we prefer to expose upright anteroposterior and lateral x-ray films of the infant if no time is lost in obtaining these studies. The operating room is prepared while these preoperative measures are in progress.

Anesthesia. The type of anesthesia which one uses will depend on the facilities under which one is working. We prefer to administer oxygen, nitrous oxide and cyclopropane through an endotracheal tube be-

cause respiration can be controlled and an adequate airway for oxygenation of the blood is assured.

TECHNIQUE. The skin of the abdomen and the sac are prepared with half-strength tincture of iodine,[41] and careful evaluation is made of the deformity. If the abdominal wall defect is small enough and the displaced viscera can be replaced into the abdominal cavity without creating too great intra-abdominal crowding and pressure, the sac is excised, the abdomen explored for other anomalies, and a layered vertical closure is performed as described by Gross.[41] Occasionally it will be necessary to enlarge the defect of the abdominal wall in order to replace the viscera.[18]

When the defect in the abdominal wall is large, most of the intestine and other viscera are contained in the sac outside the abdominal cavity. The abdominal cavity itself is underdeveloped because it has never grown to accommodate the viscera. When the liver is present in the sac, the thoracic cage is also flat and underdeveloped (Fig. 64), and the abdominal cavity is not large enough to contain the viscera. It is dangerous to crowd the viscera into the abdomen and close the fascia under tension. Four serious complications may result.[41] First, the diaphragm may be displaced upward so far that severe and fatal respira-

Fig. 64. Lateral roentgenogram of an 8-year-old boy who was born with a large omphalocele. Repair consisted in removal of omphalocele membrane, and viscera were covered with large skin flaps. Patient was referred at 2 years of age. All attempts to reduce the viscera into the abdominal cavity had failed.

tory distress and cyanosis result. Second, increased intra-abdominal pressure may hinder the return of blood to the heart from the inferior vena cava and portal vein and cause circulatory collapse and congestive edema of the legs. Third, the pressure on the stomach and intestines may lead to necrosis and perforation or obstruction. Fourth, any wound which is closed under great tension is apt to break down, and evisceration may occur.

Under these circumstances it is far better to leave the sac intact and cover it with skin flaps only. This technique was first described by Williams[102] in 1930 and was first used successfully by Gross.[40] The stump of the cord is first removed, and the umbilical vessels are individually ligated. The skin is freed from the edge of the sac and is undermined laterally as far as is necessary so as to obtain its approximation in the midline without undue tension. Gross[41] cautions against undermining the skin over the chest wall and thereby creating a subcutaneous pocket into which the liver may herniate (Fig. 64) anterior to the thorax. Closures performed in this manner heal well, and the infant may be discharged in 10 to 14 days. This method has the disadvantages that the abdomen cannot be explored for other anomalies, and it creates a large ventral hernia. The treatment of the resulting hernia (Fig. 64) is discussed below.

If the omphalocele has ruptured or the sac must be opened to correct other anomalies, closure of the skin alone results in the formation of adhesions between the viscera and the raw under surface of the skin flaps. This complicates secondary repair of the resulting hernia.

It is often difficult to replace the engorged, displaced liver which is present in the omphalocele in approximately one third of the cases.[25] Fieshman (quoted by East[29]) has been successful in replacing the engorged liver after gently squeezing it for several minutes to empty it of excess blood.

Secondary repair of the huge ventral hernia which results from a primary skin closure poses many difficult problems. Binders of various types have been used in an attempt to keep the viscera partially reduced into the abdominal cavity and to force it to grow and accomodate them. Secondary closure of the defect should not be attempted until it is believed that adequate enlargement of the abdominal cavity has occurred.

Under endotracheal anesthesia the skin flaps are then dissected from the sac, and the borders of the rectus muscle are freshened. When possible, they are approximated throughout the length of the defect with interrupted nonabsorbable sutures after the redundant sac has been removed. Often it is not possible to reduce the entire mass and close the length of the defect, and the closure must be accomplished in stages.

In 1951 Welch[100] reported the successful use of a homograft to cover temporarily a large omphalocele. The infant was 58 hours old at the time of operation, and the sac was dry and infected. The protection afforded to the infant by the homograft from the father persisted for one month. During this period a split-thickness graft from the infant and scar tissue formed an adequate permanent protective covering of the sac.*

* Editor's Note. The patient expired under my later care because of a plastic peritonitis which virtually obliterated any peritoneal cavity.—C.E.K.

Conservative therapy in the treatment of omphalocele was used by Ahlfield[25] in 1899. He placed the newborn infant under light narcosis and replaced the viscera as much as possible. Alcohol sponges and a pressure binder were then applied. Cunningham[25] reports two cases which he treated successfully in a similar manner. His illustrations demonstrate that the sac gradually becomes thickened and scarred and contracts, pushing the viscera into the abdominal cavity. The resulting abdominal wall defect is small enough to permit simple secondary closure, and reduction of the viscera is automatically accomplished by this method. A similar approach has been described by Grob[97] and Ehrenpreis.[31] They have painted the sac of the omphalocele with 2 per cent Mercurochrome daily for 3 days. The scarring and shrinking which result ultimately leave a small flat defect in the abdominal wall which can be readily closed at the time of secondary repair. This method has the disadvantage that it requires several months before healing is complete, but it may fill an important need in the treatment of omphaloceles complicated by severe infection, tremendous size, exstrophy of the bladder, and so forth.

Results. The results of therapy for the correction of omphalocele depend on several factors. In any large series of cases some of the patients will have other congenital anomalies which are incompatible with life. The size of the defect bears a direct relation to the survival of the infant. In the group of 78 patients treated by Gross at Boston Children's Hospital there was a 75 per cent survival of those infants whose abdominal wall defect was less than 7 cm. in diameter, and only a 15 per cent survival of those in whom the defect was over 9 cm. in diameter.[41]

The liver is included in the sac in 33 per cent[29, 41] to 50 per cent[37, 63] of patients with omphalocele. Its presence decreases the chances for the infant's survival. If rupture of the sac has occurred, the prognosis for recovery is decreased greatly. Burgess,[16] however, states that one third of the patients with ruptured omphalocele reported in the literature have survived. In Gross's[41] series 6 of 17 patients with ruptured omphalocele survived, and in McKeown's series[66] the survival rate was 7 of twenty-seven.

The length of time between birth of the infant and treatment of the omphalocele is one controllable factor of great importance. East[29] quotes Alder, who found a mortality rate of 12 per cent in patients operated upon at less than 12 hours of age, and of 66 per cent in those operated upon after 48 hours of age. Kern[62] reports a mortality of 21.4 per cent of patients under 12 hours of age, 44.4 per cent in those 24 hours of age and 61.6 per cent in those over 24 hours of age.

The existing over-all survival rate in large series of infants who have been operated upon for omphalocele ranges from 62 per cent of 78 patients[41] to 76 per cent of 91 patients.[63] With the development of modern surgical and anesthetic techniques, antibiotics and fluid therapy, these survival figures should be increased if the importance of immediate therapy is appreciated, since the percentage of patients with other anomalies which are incompatible with life is relatively small.

EXSTROPHY OF THE BLADDER; EXSTROPHY OF THE CLOACA; GASTROSCHISIS

Other somatic anomalies which occur about the umbilicus include such conditions as exstrophy of the bladder, exstrophy of the cloaca, and gastroschisis. Exstrophy of the bladder may be accompanied by an umbilical hernia or omphalocele, which further complicates therapy. It is not within the scope of this paper to discuss this complex anomaly further. (See also p. 998.)

Exstrophy of the cloaca[96] and vesicointestinal fistula[42] are terms used to describe a rare combination of multiple anomalies of the abdominal wall. There is usually a bifid exstrophy of the bladder and often biphallus with separate urethras joining the bladder. Between or adjacent to the bladder segments two intestinal openings are present. One of these drains the terminal ileum, sometimes through a Meckel's diverticulum. The other opening leads to the cecum, and the colon always ends blindly within the abdominal cavity. An omphalocele is frequently present and is separated from the exstrophied bladder by a narrow band of skin. Multiple other anomalies are also usually present. It may be possible to reconstruct some of these defects satisfactorily, but attempts to do so by Swan[96] and ourselves have not succeeded to date.

Gastroschisis means "belly cleft"[64] and denotes a localized congenital complete defect of the abdominal wall lateral to the umbilicus. Strangulated loops of intestine[64, 72, 75] or omentum[103] protrude through the defect at birth, and there is no protective sac. The umbilicus is normal. In 1957 Kiesewetter[64] reported the tenth case in the literature. The salvage of some of these infants is possible, depending upon the amount of intestine which is viable.[75]

EPIGASTRIC HERNIA

Epigastric hernias occur through defects of the abdominal fascia in the midline superior to the umbilicus. In contrast to the usually asymptomatic umbilical hernia, these small defects frequently cause symptoms due to protrusion of a portion of the omentum within them. The defect will not regress spontaneously, and surgical repair should be undertaken when one is discovered. We use a transverse skin incision and a vertical fascial closure using stainless steel wire.

DIASTASIS RECTI

Diastasis recti is frequently present in conjunction with an umbilical hernia. When an umbilical hernia is repaired, using a vertical closure,

the diastasis recti may be approximated at the same time. Repair of the defect when it is an isolated condition is rarely if ever indicated.

ACQUIRED LESIONS OF THE UMBILICUS

INFECTIONS

The importance of using aseptic techniques in severing the umbilical cord cannot be overemphasized. A recent report from Russia[78] states that 9.6 per cent of deaths in children under 1 year of age were due to omphalitis, 65 per cent of which were not recognized clinically. Two recent case records from Massachusetts General Hospital[69, 70] emphasize the seriousness of this condition.

Runge is given credit by Cullen[24] for being the first to emphasize the importance of a dry cord in preventing infections, and Hardyment[43] in 1958 re-emphasized the adverse effects of moisture. He quotes Jellard, who recommends that the stump be painted daily with triple dye to decrease the incidence of staphylococcal infection (see recommendations of the American Academy of Pediatrics).

"In every instance of illness in a newborn infant it should always be a rule to inspect and, if necessary, re-inspect the navel." Cullen[24] made this statement after he had studied many detailed reports of omphalitis, and recent articles confirm the astuteness of this observation.[11, 26, 52, 69, 70] The infection may present as a frank umbilical abscess with localized gangrene or slough of varying amounts of the abdominal wall. A more common and more insidious form which invades through the umbilical vessels may be manifest solely by the infant's fretfulness, irritability or lassitude and refusal to feed. This type of omphalitis may occur many days after the cord has dropped off and the umbilicus appears to be clean and dry.

Studies have shown that the umbilical vessels are the usual source of the infection.[24, 51] Some of the complications in neonatal omphalitis include jaundice, hepatic abscesses,[52, 82] thrombosis of the portal vein,[69, 94] pyelonephritis,[26] meningitis,[70] pneumonia,[24] peritonitis[24, 49, 69] and septic arthritis.[24, 51] Recent outbreaks of infection in mothers and aides of maternity units have been traced to infections of the umbilicus of the infants whose umbilici appeared grossly normal.[11, 65] In the past, streptococci were the most frequently isolated pathogenic bacteria, but Burgin[70] and Isbister[52] found that staphylococci are now the most common offenders. Diphtheria, syphilis, and tetanus of the umbilicus were reported frequently in the older literature[24, 51] and probably are still seen in areas in the world where asepsis is not practiced.[82]

As we have indicated, omphalitis may have severe sequelae both to the infant and to those who care for newborn infants. With an in-

creasing number of pathogenic bacteria developing strains resistant to chemotherapy, it is essential that strict asepsis be practiced in treating the umbilical cord. The stump should be closely examined as a possible portal of entry for infection in every newborn infant, since the umbilical vein may not close for three to four weeks.[26, 76]

GRANULOMA

Delayed healing of the umbilical stump accompanied by formation of granulation tissue is the most common disease of the umbilicus of the newborn. Mild infection is probably the cause.[51] Treatment consists in cauterization with silver nitrate and the application of alcohol sponges. If there is recurrence of the granuloma, the persistence of urachal or omphalomesenteric duct remnants should be considered (see Table 1).

HEMORRHAGIC LESIONS

Severe hemorrhage from the umbilical stump is surprisingly rare. The tie or clamp rarely slips off. A generalized disturbance of the infant's ability to coagulate its blood should be suspected if a significant amount of bleeding occurs. Control of the bleeding is best accomplished by transfusion of whole fresh blood. Local pressure on the stump may be helpful, and attempts at retying and electrocoagulation are sometimes successful. Hemorrhage from the umbilical stump may occur after the cord has separated if a persistent omphalomesenteric duct with its accompanying artery is present.

TRAUMATIC LESIONS

Infants and children who have umbilical hernias are apt to investigate them with pencils, sticks, and the like. The skin may become irritated from pulling at the hernia, especially if it protrudes much (Fig. 60).

CONCRETIONS

The formation of umbilical concretions is rare in children. They are formed from dirt, debris from clothing and desquamated epithelial elements which collect in a deep umbilicus with a narrow external opening. In adults Cullen[24] records instances in which the concretions reached the size of a hen's egg. The authors have seen one patient who complained that odoriferous cheesy material collected in his umbilicus. The patient was an obese boy, 10 years of age, whose umbili-

cus had a narrow external opening which led to a small pocket. Thorough cleansing of the tract at regular intervals relieved the complaint.

TUMORS OF THE UMBILICAL AREA

Tumors of the umbilicus and umbilical area are rare, and in infants and children are usually benign. Malignant lesions usually appear later in life, but have been reported in childhood.[24]

A complete classification of the tumors of this area is given by Cullen;[24] many of these are observed primarily in adult life, however. It is apparent that almost any tumor of soft tissue origin may arise in this area, but only a few of these lesions which may be encountered in early life will be discussed briefly.

Granulomas, which are the most frequent tumors in this site, are inflammatory rather than neoplastic. The differential diagnosis between these and umbilical polyps is recorded on page 1092.

Angiomas of the anterior abdominal wall may have capillary or cavernous elements or may be mixed. Those with cavernous elements tend to penetrate into the abdominal wall. Large superficial vessels often communicate with these tumors. Owing to the mass of dilated vessels, the tumor has a bluish discoloration. These deep-seated vascular tumors should be totally excised.

Capillary hemangiomas have an irregular, flattened strawberry appearance. These tumors usually disappear spontaneously, and therapy is rarely necessary.

Lipomas of the abdominal wall are not common. Those which are encapsulated are easily removed, and recurrence is unlikely. Other lipomas which are not encapsulated (diffuse lipomatosis) and which invade the muscle are notorious for their recurrences; these, however, probably represent hamartomatous malformations rather than true neoplasms.

Desmoid tumors usually occur inferior to the umbilicus in the rectus fascia and muscle in infants and children;[41] they may, however, be observed in other sites.

We have encountered an infant, 4 weeks of age, who presented with a small mass in the rectus fascia and muscle just inferior to the umbilicus. At operation a poorly circumscribed fibrous mass was widely excised with the removal of adjacent fascia and muscle. Recent follow-up, 7 years after excision of this mass, revealed no evidence of recurrence.

These tumors are, however, notorious for their high incidence of recurrence. Wide excision should be carried out with removal of normal tissue adjacent to the lesion, and in spite of this repeated recurrences may ensue.

Teratomas are rare, but do occur in the abdominal wall. Gross[41]

reports one patient with such a tumor who died of other causes after excision of the tumor.

Dermoids of the umbilical area may be large or small. They are cystic structures which contain sebaceous material. The overlying skin is usually unaltered, but may be thin, depending upon the size of the tumor. Surgical excision, including removal of the capsule, is recommended.

Malignant tumors of the umbilical area are rare in patients under 25 years of age. One such case was encountered in reviewing the literature. A patient 11 years of age, reported by Cullen,[24] had a sarcoma develop in the umbilicus.

The incidence of adenocarcinoma from urachal remnants in adulthood stresses the importance of diagnosis and removal of these elements in childhood.

SUMMARY

The diagnosis and treatment of congenital and acquired lesions which occur in the region of the umbilicus of children have been discussed. A summary of the embryologic development of the umbilical area has been presented.

The recognition and treatment of alimentary tract and urachal anomalies are important because some of the complications due to their persistence may be lethal. We believe that umbilical hernias which have a persistent defect in the fascia of more than 2 cm. by the time the child is 1 year of age should be repaired surgically.

It is essential to evaluate infants born with omphaloceles for other anomalies. Immediate surgical repair of the omphalocele is the recommended treatment. A nonoperative method of therapy is discussed.

The importance of using aseptic technique for the care of the umbilical cord is emphasized. Infection of the umbilicus in the newborn infant may be insidious. Unrecognized omphalitis may have severe sequelae both to the affected infants and to those who care for them.

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CHRONIC PYELONEPHRITIS MIMICKING URETERAL OBSTRUCTIONS

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Chronic or recurrent pyelonephritis frequently causes so few symptoms that the patient is unaware of his disease. This insidious characteristic plus the paucity of objective signs allows progressive renal and ureteral damage to remain unrecognized by the physician for months or years. When the disease is recognized and is treated successfully, ureteral and bladder function returns to normal as the inflammatory reaction disappears. Simultaneously hypertrophy of the remaining nephrons replaces the function of those destroyed by the infection. The end result can be a normal urinary system.

INCIDENCE

Various pediatricians claim that the problems of urinary infections occupy approximately 5 per cent of office pediatric practice and about 1 per cent of pediatric hospital admissions. Autopsy statistics indicate that pyelonephritis is frequently unrecognized by the clinician, that it is almost always associated with chronic ureteritis and chronic cystitis, that it is the most common cause of death due to renal failure, and that it kills twice the number of people as does glomerulonephritis and twice as many girls as boys. In clinical practice, however, urinary infections occur 6 to 10 times more frequently in girls than in boys.

CAUSES

Perhaps the girls' short urethra accounts for the higher frequency of urinary infections in this sex. At Stokes'[7] suggestion we were able to

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show that dyed bath water can go into the bladder of normal girls. In susceptible girls the frequency of recurrent urinary infections can be diminished by avoiding tub baths and swimming. But if tub bathing, i.e., the short urethra, is the main etiologic agent, then more girls should have urinary infections. Since this hypothesis is not true, much etiologic information is missing.

Many investigators have established the fact that when the urine flow is sluggish, a transitory septicemia can cause a urinary infection. Although most clinicians are aware of the various anatomic obstructions to urine flow, few are conscious of the nonanatomic causes of a residual urine, e.g., dysuria resulting from a urethral meatal ulcer in a recently circumcised boy and from vulvitis in the girl. The child reacts to pain on voiding by trying not to urinate and partially succeeds until the resultant bladder infection and irritability cause tetanic bladder contractions. Only recently has the clinician become conscious that urethral instrumentation necessarily carries saprophytic urethral bacteria into the bladder.[2] Thus an indwelling catheter always causes a cystitis. Originally it was thought that a cystitis might cause a pyelonephritis only if it caused a septicemia. Recent work has shown that chronic bladder infections stiffen the vesical intramural ureter, leading to vesical-ureteral-renal reflux and pyelonephritis.[1, 8] Also when the bladder is overdistended, its wall becomes thin[3] and the vesical intramural ureter becomes short, allowing vesical-ureteral-renal reflux to occur. Although many doubt the etiologic aspect of the girls' short urethra, we have no doubt that infected bladder urine can frequently cause a pyelonephritis.

PATHOLOGY

In the kidney localized collections of inflammatory cells replace tubules, cluster about glomeruli and blood vessels, and eventually cause periglomerular and perivascular fibrosis. In the infundibula, renal pelvis, ureter and bladder similar collections of inflammatory cells fill the submucosal space, infiltrate between muscle bundles and subsequently replace them. Thus by gradually destroying the urinary transport mechanism and by causing an increase in renal pelvic pressure this disease tends to perpetuate itself and to make treatment difficult.

The impaired function of the infundibula, pelves and ureters is readily visualized on the x-ray film. In Figure 65 the dilatation of the infundibula, pelvis and ureter on the left is obvious when it is compared with the normal right side.

A 7-month-old-boy* was admitted to hospital with a temperature of 102° F. in

* The author is grateful to Dr. Wallace W. McCrory, who cared for the patient and permitted us to use this patient material.

spite of varied antibiotic therapy in the previous 3 weeks. On physical examination he had blood in the left auditory canal, hyperemia of the left ear drum, a mild nasopharyngitis, a slightly enlarged left kidney, and a severe phimosis with a urethral meatal ulcer. The initial urinalysis showed only 3 to 5 white blood cells and a subsequent one revealed the same number of white cells plus 40 to 50 red blood cells per high-power microscopic field. A coliform organism was cultured from the urine. The enlarged left kidney and the hematuria prompted the pediatrician to order the intravenous urogram.

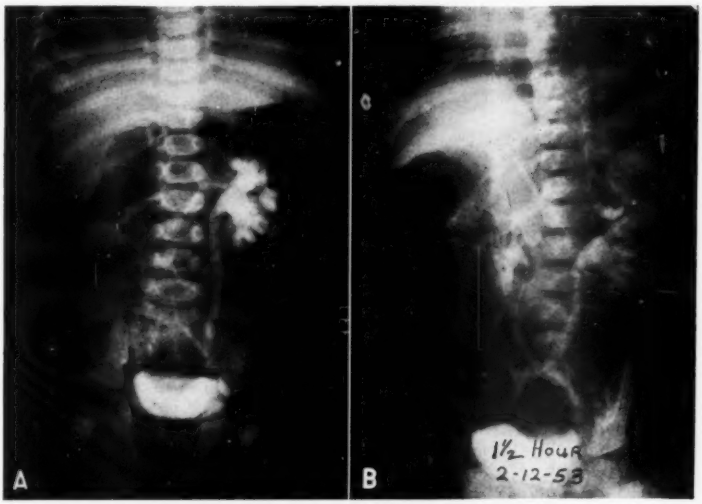

Fig. 65. A, The dilatation of the calyces, pelvis and ureter of the left kidney is obvious. There appears to be not only a ureterovesical obstruction, but also compression of the upper ureter by 3 aberrant vessels. *B*, Although the configuration of the right kidney is normal, there is an obvious anomalous dilatation of the lower two thirds of the right ureter. The dilatation of the left urinary system is again seen.

The strange dilatation of the distal two thirds, but not of the proximal right ureter, seemed to indicate that a ureteritis was extending cephalad from the bladder. But at this time we could not decide whether the patient had (*a*) bilateral ureterovesical obstructions or (*b*) faulty ureteral peristalsis and the ureteral dilatation because of chronic ureteritis and pyelonephritis. In this patient a urinary infection could have developed from 2 unrelated causes: (*a*) The urinary infection might have been a metastatic blood-borne infection from the left otitis media. (*b*) The dysuria of the urethral meatal ulcer might have caused a chronic bladder residuum which in turn led to the urinary infection and the ureteritis. Since most of the known facts indicated that the ureteral dilatation was caused by infection, a circumcision was performed and antibiotic therapy begun. In order to be certain that we were not missing an anatomic ureterovesical obstruction, the pediatrician told the parents that he wanted to repeat the intravenous urogram in 3 months. After a month of antibiotic therapy, however, the baby was so well that the parents stopped the treatment and decided not to bring him back for the repeat intravenous urogram. But 18 months later, when their child became hyperirritable, he was brought back to the hospital. At that time all studies, including the repeat intravenous urogram (Fig. 66), were normal. Thus the ureteral and calycine dilatation in this patient was due to the urinary infection and disappeared when it was eradicated.

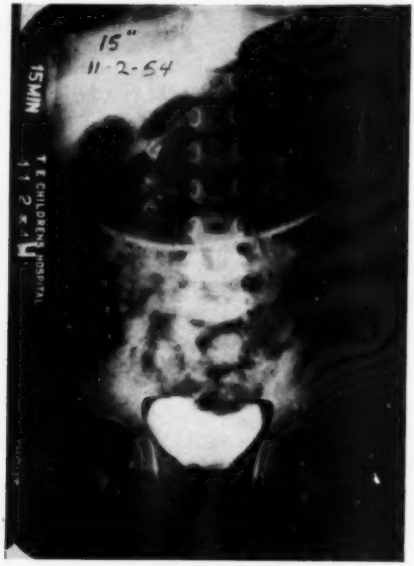

Fig. 66. About 17 months after a circumcision and the cessation of sulfonamide therapy, the dilatation of the ureters and of the pelvis and calyces of the left kidney has gone.

Fig. 67. Apparently bilateral ureterovesical obstructions have caused the striking dilatation of the upper urinary system in this 2-month-old baby girl.

A similar but more exaggerated renal pelvic and ureteral dilatation occurred in a 2-month baby girl. The intravenous urogram (Fig. 67) showed blunting of all calycine pyramidal angles and dilated pelves and ureters. These changes were greater on the right side and were associated with a large bladder which did not completely empty itself. The baby had been completely asymptomatic until she was 2 months old, when she suddenly had a temperature of 105° F. A urinalysis revealed innumerable white cells, and Proteus grew in the cultured urine. At this point the diagnosis of acute and chronic pyelonephritis, ureteritis and cystitis was obvious. The possibility that this baby might also have a bladder neck obstruction or bilateral ureterovesical obstruction was considered. But the severe fever forced us to treat the infection first.

Initially, with little success, various antibiotics were given singly. Later a combination of an antibiotic and a sulfonamide at first reduced and later stopped the recurrent fevers in 5 to 6 months. At this time the antibiotic was stopped, but the sulfonamide therapy was and has been continued up to the present, since Proteus is still growing in current urine cultures. The pyuria disappeared one year after starting the sulfonamide therapy. Serial intravenous urograms showed that the blunting of the calycine pyramidal angles and the dilatation of the pelves and ureters gradually returned to normal (Fig. 68)

Fig. 68. The dilatation of the upper urinary system seen in Figure 67 has gone 16 months after the institution of antibacterial therapy. No operation was performed.

16 months after starting the antibacterial treatment. Now the bladder can empty itself completely. Thus, in this patient also, a urinary infection and the resultant inflammatory reaction caused the hydronephrosis, the hydroureter and the poorly functioning bladder.

DIAGNOSIS

Since the patient rarely complains of symptoms specifically referable to his urinary infection, the diagnosis rests on the results obtained from

satisfactory urine cultures. Infected urine from the kidneys is stored in the bladder. Consequently a varying degree of cystitis is present in all patients with pyelonephritis, yet symptoms of bladder irritability such as urinary frequency, urgency, incontinence or dysuria occurred in only 60 per cent of our patients. Symptoms of a transitory septicemia often bring the patient to the pediatrician and the hospital, but this occurred in only 20 per cent of our patients with proved recurrent pyelonephritis. Pyuria, more than 20 white blood cells per high-power field in the sediment of a centrifuged urine, occurs in about 90 per cent of the untreated patients. Although vaginal secretions in the girl and preputial secretions in the uncircumcised boy can produce confusing degrees of pyuria, this diagnostic difficulty can be avoided technically. However, since pyuria can occur when the urine culture is sterile (Table 2), the only reliable diagnostic test is the repeated demonstration of the same bacteria in repeat urine cultures.

Falsely positive urine cultures occur frequently because saprophytic bacteria normally live in the urethra between the urethral meatus and the voluntary urethral sphincter. Although transurethral instrumentation of the bladder always carries saprophytic urethral organisms into the bladder, a week later no significant pyuria is present, and the urine cultures are usually sterile except when an indwelling urethral catheter has been in place for more than 48 hours. Although many physicians believe that a representative sample of bladder urine can be obtained through a urethral catheter, falsely positive cultures also are readily obtained,[6] for the eyes of the catheter in passing through the urethra scrape the saprophytic organisms into the catheter lumen.

The true bacterial status of the urine can be determined by culture of the urine aspirated from the bladder through a suprapubic needle,[4] but it is not necessary to do this. From classic hydraulics we know that when fluid flows in a straight pipe at a constant rate, an immobile layer of fluid covers the lining of the pipe, e.g., the contaminated lining of the inserted urethral catheter or the infected surface of the urethral mucosa. Accordingly, in girls a satisfactory urine culture is obtained from a catheter only when the sample is caught in *mid-stream* when the rate of *urine flow* is *constant*. Although the male urethra is not straight, its bend is sufficiently gentle so that the resulting turbulence does not prevent a procurement of sterile *mid-stream* urine cultures in normal boys. Gross turbulence of the urethral urinary stream can be avoided in boys by retraction of the foreskin and in girls by spreading of the labia.* With this technique a satisfactory *mid-stream* urine

* Because it is psychologically difficult for girls to cooperate with this technique, only a few will do it satisfactorily. Thus urethral catheterization is used to obtain urine cultures in most of our female patients.

culture can be obtained from either sex without catheterization or suprapubic aspiration.

By using the *mid-stream* technique we obtain sterile cultures from normal subjects in spite of the fact that 2 ml. of their urine are incubated for 5 days. We assume that, in the absence of a septicemia, the urine formed by the kidneys is bacteriologically sterile. Conversely, we cannot accept the tenet that normal urine contains a definite number of viable bacteria. This finding by other investigators[5] can be explained by contamination of sterile normal urine with varying degrees of saprophytic urethral organisms. By the use of the *mid-stream* technique and by incubating 2 ml. of the obtained urine sample in a meat broth tube for 5 days, the incidence of falsely negative cultures is minimal, but the frequency of falsely positive cultures is maximal. Because more than one species of bacteria lives within the urethra, it is unlikely that the same bacterial species will grow from two or three successive urine cultures. Accordingly, if two or three urine cultures reveal the same bacterial species, the patient has a urinary infection. Likewise, if a single urine culture is sterile, the patient does not have a urinary infection.

TREATMENT

In the care of a group of patients with pyelonephritis there is a common basic treatment to which is added a variable supplementary treatment for each patient. In attempting to formulate a common basic treatment for chronic pyelonephritis, the author reviewed his previous experience and noted that the patients' reaction to treatment can be utilized diagnostically. By using a different basis of treatment the author is able to reduce appreciably the number of intravenous urograms and urine cultures required to treat the patient with chronic pyelonephritis satisfactorily.

The suggested basis of treatment (Table 2) has worked and is working satisfactorily in our clinics. The number of intravenous urograms, urine cultures, and visits to the doctor's office are reduced to a minimum, but the patient is overtreated. However, unless the patient has a severe toxic reaction to a drug, the cost of the drugs rarely equals the cost of the intravenous urograms and the urine cultures which we formerly required to treat our patients and which were required to evaluate our current basic treatment program. During the initial phase of the sulfonamide treatment it is important to tell the patient to return to his physician if his symptoms do not subside and disappear in three to five days. By continuing the sulfonamide therapy after obtaining a sterile urine culture we reduce our recurrence rate from 20

TABLE 2. *Treatment of Pyelonephritis*

FIRST PHASE

Urinalysis....Pyuria: More than 20 white blood cells per high-power field in a centrifuged urine sediment
Symptoms....Urinary frequency, dysuria, urgency, incontinence, chills, fever, abdominal pain, nausea and/or vomiting
Findings....Vague suprapubic tenderness and rarely costovertebral angle tenderness
Impression....Urinary infection. When a fever is present, a pyelonephritis probably exists
Treatment....To reduce the incidence of drug resistance by the bacteria, the treatment must be continuous. One drug is given until the next is picked. Start with a soluble or triple sulfonamide, e.g., sulfisoxazole, sulfisomidine, etc. Dosage: 0.1 gm./lb./day given in 3 equal doses at 8-hour intervals for 4 weeks. Then a repeat urinalysis and a urine culture enable you to place the patient in one of 4 groups:

SECOND PHASE

Urinalysis	Pyuria	Pyuria	No pyuria	No pyuria
Urine culture	Sterile	Positive	Sterile	Positive
Symptoms	Unchanged or asymptomatic	Usually asymptomatic	Asymptomatic	Asymptomatic
Findings	No change	Previous tenderness persists	None	None
Diagnosis	1. Glomerulonephritis 2. Unusual renal diseases: Renal tuberculosis Disseminated lupus Sarcoid Leukemia Lymphoma Multiple myeloma	An obstruction to urine flow is present	1. Cystitis 2. Early minimal pyelonephritis	Chronic pyelonephritis
Treatment	Depends on establishment of the correct diagnosis	1. Surgical consultation 2. Intravenous urogram	1. Reduce the sulfonamide therapy by one third; i.e., give the same individual dose at 12-hr. rather than 8-hr. intervals 2. Continue this treatment for 3 months, and then stop 3. Obtain 3 repeat urine cultures at 3-month intervals after treat-	1. Continue the sulfonamide treatment and repeat the urine cultures at weekly intervals until 2 or 3 consecutive urine cultures show the same organisms. Then: 2. Give the appropriate antibiotic as indicated by the bacterial sensitivity. Dosage: 20 mg./lb./day until the patient weighs 100 lbs. The drug is given in 3 equal doses at 8-hr. intervals 3. Repeat the urine culture in 2 weeks

	and then stop	3. Repeat the urine culture in 2 weeks
	3. Obtain 3 repeat urine cultures at 3-month intervals after treatment has been stopped. If these are sterile, we believe the patient is cured	

THIRD PHASE

Urine culture	Sterile	Positive
Treatment	1. For the first 6 months give the full dosage of a soluble sulfonamide, 0.1 gm./lb./day in 3 equal doses at 8-hr. intervals. Then repeat the urine culture. If sterile, then: 2. For the second 6 months give two thirds of the original dose, by giving the same individual dose at 12-hr. instead of 8-hr. intervals. If the urine culture at the end of this period is sterile, 3. For the third 6 months reduce the sulfonamide dosage of the second period by a half; i.e., give half the dosage of the second period at 12-hr. intervals. Then repeat the urine culture. If sterile, 4. Stop the treatment and obtain 3 repeat urine cultures at 3-month intervals. If these are sterile, we believe the patient is cured	1. If the bacteria have changed, repeat the urine culture 2. If the same bacteria and drug sensitivities are present, continue the same antibiotic for another 2 weeks and then repeat the urine culture 3. If the same bacteria are present, but the drug sensitivities are different, pick another antibiotic and give it for 2 weeks and then repeat the urine culture. If sterile, 4. Treat as if the culture became sterile in 2 instead of 4 weeks of antibiotic therapy 5. If the culture is positive, try other drugs and combinations

to less than 5 per cent. After cessation of therapy it is important to obtain at least three successive urine cultures at three-month intervals because of the high incidence of asymptomatic pyelonephritis. Occasionally it has taken 2 to 3 years to eradicate a urinary infection, but about 80 to 90 per cent of our patients respond to the treatment program outlined in Table 2.

In our practice there are a group of girls who have recurrent urinary infections in spite of the prolonged therapy. These patients lose this susceptibility shortly after the onset of the menstrual cycle. We have induced periodic vaginal bleeding for a few months in two such girls aged seven and nine years. However, such endocrine therapy did not prevent a recurrence of their urinary infection. Now both patients have well established menstrual cycles and no longer have recurrent urinary infections. Urethral length may explain the difference; in the girl it is 1 to 3 cm. long, and in the woman 5 to 8 cm. long.

Many pediatricians ask me whether the treatment is worth while, since many patients are asymptomatic with their disease. In a few errant patients studied, chronic minimal pyelonephritis diminishes the glomerular filtration rate and the maximal tubular excretion of p-aminohuppurate 10 per cent of what remains each year. By extrapolation, if both kidneys are diseased, the total renal function will be less than 10 per cent of normal in 25 to 35 years. If only one kidney is affected, compensatory hypertrophy of the nephrons in the contralateral kidney increases its function proportionally so that there is no reduction in total renal function. Also in a few cases studied if treatment is successful in children with bilaterally diseased kidneys before the total renal function is less than 10 per cent of normal, hypertrophy of the remaining nephrons almost completely replaced the function destroyed by this disease.

SUMMARY

In the early part of the twentieth century Young[9] established the doctrine that when a dilatation occurs in the urinary system, there is an anatomic obstruction to urine flow distal to the dilatation. According to this precept, successful treatment requires the surgeon to find and to remove the obstruction. In practice it is not always possible to demonstrate the obstruction by x-ray study or by operation. If the obstruction is a paralysis of the propulsive function of the ureter caused by ureteritis, no anatomic obstruction will be found. Young could not, but current physicians can eradicate an infection in a dilated urinary system with antibiotics. Thus we are able to link the effect of infection on ureteral function. In the middle of the twentieth century Young's precept must be changed:

1. First attempt to eradicate the urinary infection and then re-evaluate the dilatation.

2. If the dilatation of the urinary system remains, operate and remove the anatomic obstruction. Or,

3. If eradication of the infection is impossible, surgical exploration is warranted.

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HERNIAS AND HYDROCELES

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The subject of inguinal hernias and hydroceles may well be considered as a unit, since most hydroceles are simply manifestations of a basic hernia-hydrocele complex. Except during the first three or four months of life, when a hydrocele may be physiological, one is safest in assuming that any hydrocele in a child requires elimination of the accompanying hernia as the underlying cause.

EMBRYOLOGY

A clear understanding of the embryologic development of the inguinal and scrotal areas does much to clarify the understanding of a hernia-hydrocele.

The testicle is an extraperitoneal and abdominal organ in the process of its early development. At approximately the sixth to seventh month of gestation (Fig. 69, *A*) a small outpouching of the peritoneum proceeds through the internal inguinal ring, down along the inguinal canal, out the external ring, and into the bottom of the scrotum. This outpouching is referred to as the processus vaginalis. The testicle then descends through the previously opened inguinal canal and scrotum to its final resting place in the lower part of the sac (Fig. 69, *B*). At this point the processus vaginalis closes off above the testicle from a point in the scrotum up to the internal inguinal ring (Fig. 69, *C*). However, there is left behind a small vestige of the processus vaginalis around the testicle, which contains a small amount of fluid giving mobility and

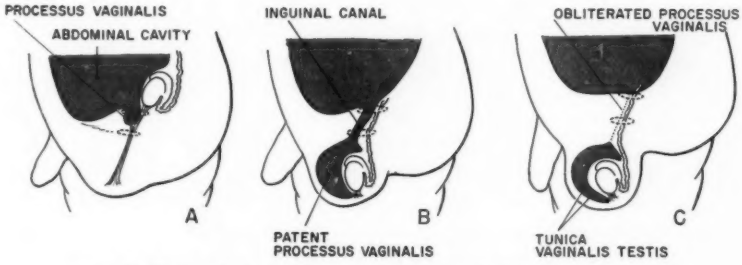

Fig. 69. Embryologic development of the inguinal and scrotal areas.

cushioning to the testicle itself. This remnant is called the tunica vaginalis testis. It has been estimated that the processus vaginalis on the left closes about 3 weeks before term, and that closure on the right follows about 1 week later.

PATHOGENESIS

On the basis of the embryologic development of the inguinal and scrotal areas, there are two ways by which a hernia-hydrocele can arise: (1) A child who has undergone a normal and complete closure of its processus vaginalis may, under a period of stress, with increased intra-abdominal pressure, wedge the processus open once again. By this increased pressure of coughing, crying or straining the previously obliterated communication between the abdominal cavity and the inguinal-scrotal area is re-established. (2) Some children never completely obliterate the processus vaginalis and are therefore born with an already persisting communication between the abdominal cavity and the inguinal-scrotal area. Since closure of the processus vaginalis occurs late in pregnancy, the frequency of hernia-hydrocele in premature children can be readily understood.

DIAGNOSIS

Types

In Figure 70 we have diagrammatic representations of the four classic types of hernia-hydrocele that are encountered.

In Figure 70, *A*, we have represented an inguinal bulge, which is frequently the first evidence parents have that the child is suffering from a hernia. It portrays a communication between the inguinal canal and the peritoneal cavity. The parent's story is usually that of noticing a "lump in the groin" when the child is being bathed or dressed. It is

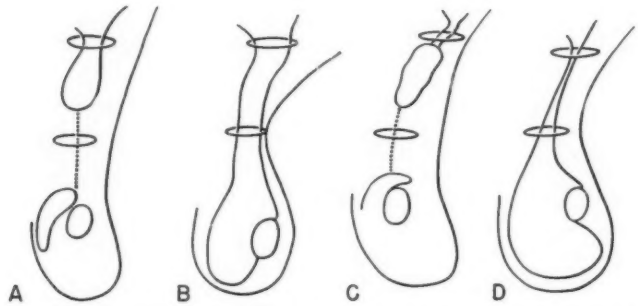

Fig. 70. Diagrammatic representation of the 4 clinical manifestations of a hernia-hydrocele: *A*, the inguinal bulge; *B*, the scrotal bulge; *C*, the hydrocele of the cord; *D*, the scrotal hydrocele.

reproducible by having the older child stand up or increase his intra-abdominal pressure by coughing or "bearing down." In the smaller child this is often involuntarily achieved by crying during examination.

Figure 70, *B*, indicates diagrammatically the further progress of the inguinal bulge by showing that the processus vaginalis is completely open between the abdominal cavity and the bottom of the scrotum. This is manifested to the family as a large inguinal-scrotal bulge. This bulge may be due to fluid or intestines, or a combination of the two. The history the family gives of such a child is that a small inguinal bulge has gradually, over a period of weeks or months, become larger and larger until its very size has forced them to seek advice. Although not the commonest variety of hernia, it is by all odds the easiest to diagnose. Placing such a child in a recumbent position will allow easy reduction of the hernia and confirm one's diagnostic impression.

Figure 70, *C*, is a representation of the most difficult type of hernia-hydrocele to be sure of. It is called by various names, the most common being "hydrocele of the cord." In the female this even rarer manifestation is called a "hydrocele of the canal of Nuck." The diagrammatic sketch shows clearly that the processus vaginalis is patent for a varying distance down towards the pubic tubercle and that intraperitoneal fluid has passed through this patency. It becomes sealed off at its proximal end so that the fluid does not readily return to the abdominal cavity. These lumps are generally almond-sized, and the family's story is that it suddenly appeared without any pain or symptomatology. They have already palpated it thoroughly and will tell you that it moves readily without any apparent concern on the part of the child. The history may also reveal that this lump has come and gone on several occasions, each time without any obvious distress to the patient. This is the most difficult type of hernia to be sure of, and its import is obvious. If it is only a hydrocele of the cord, there is no intestine complicating the picture, and correction is of elective significance. If there is bowel involvement, however, this represents an emergency situation. Helpful points of distinction are that the mass is freely movable and that moving it toward the pubic tubercle allows one to place his finger over a normal area of the inguinal canal between the mass and the internal inguinal ring. In very small infants it is possible, by doing a rectal examination, to feel the internal ring from the inside and thereby determine whether bowel is going through it or not.

Figure 70, *D*, depicts the final type of evidence which one may get of the presence of a hernia-hydrocele. It is the most confusing variety, and the one about which there is the greatest amount of controversy. The schematic representation shows that there is a narrow channel between the abdominal cavity and the scrotum, through the inguinal canal. The manifestation of this communication is all scrotal, in the

form of accumulation of fluid, called a hydrocele. The very presence of this fluid in the scrotum is evidence of the presently existing, or immediately preceding, communication between the hydrocele cavity and the abdomen through the associated hernia. The story given by the family is once again that of an asymptomatic, increasingly large lump in the scrotum. They may also give a story of fluctuation, indicating that the size of the mass is much smaller in the morning after a period of horizontal rest. That is important in that it indicates that there is a patency (a hernia) by which fluid can flow in both directions.

In summary, then, the clinical types of hernia-hydrocele manifestation that one encounters include two in which a hernial sac, with intestinal contents, is present and two in which the presence of the sac is indicated by an accumulation of intra-abdominal fluid which has passed down through the patent processus vaginalis.

Physical Examination

In conjunction with the above-mentioned history of hernia-hydrocele, there are also at least three additional steps in physical examination which will help make the diagnosis of the condition. The first of these is the observation of the two inguinal areas to see whether there is any relative fullness or protuberance on one side as opposed to the other. This is much less reliable in the well nourished child, who often has a considerable amount of fatty deposit in this area. Secondly, one examines the inguinal and scrotal areas to see whether there is any thickness in the cord structures on one side as opposed to the other. Finally, one has the ability to palpate an unusual crepitation in the area of the internal ring which has often been referred to as the "silk sign." This is thought to represent the ability to rub the two sides of the sac together and is confirmatory, if present, of the presence of a hernia. This palpation, with one finger, should be carried on away from the pubic tubercle, where one can often be confused by moving normal cord structures against the bone and get the same sensation.

COMPLICATIONS

From Erroneous Concepts

In thinking through the problems presented by a hernia-hydrocele, at least four misconceptions appear with some frequency. Parents are often told that their child's hernia "will go away." No one can deny that there are certain hernia-hydrocele manifestations that do seem to vanish. It would be most unwise, however, from a practical standpoint, to give this type of reassurance to every parent, because only a very

small percentage of patients manage their condition in such an expert, spontaneous fashion. If one is going to be selective in the use of this advice (which we think is seldom indicated), it would appear best to be reserved for those children with a simple hydrocele in the early months of life. The second misconception is that the hernia "will recur" if operated on too early. Because we have had the opportunity to observe the results in close to 4000 herniorrhaphies, this does not seem to be valid advice. There have been less than 12 recurrences in this large group of inguinal hernia repairs, and most of the herniaplasties were done in the early months and years of life. A third piece of advice that is often given to parents is that the child is "too young" for surgery. This too would seem to be based on other considerations, such as anesthesia inadequacies, rather than upon the age of the patient. Given proper ancillary help, a surgeon used to working in the inguinal area of an infant can do the job as effectively at an early age as later. Finally, there has been in recent writings[3] a suggestion that hernias in female patients are better handled in a somewhat different way than in males, in view of the danger of involvement of the internal genitalia. This deferment of surgical correction until an older age in females does not seem to be based on sound concepts. With proper care to the internal organs which may be attached to the sac, correction of such hernias can be accomplished with less risk than in the male because of the lack of a spermatic cord adjoining the hernia sac.

From the Disease

The real concern in the mind of every physician who sees a hernia-hydrocele situation is the fear of incarceration. This has been variously reported as occurring in 5 to 15 per cent of cases.[2] Not all incarcerations lead to emergency operation, because many reduce themselves spontaneously under sedation or an automobile ride to the hospital. All observers agree, however, that the incidence of incarceration is greatly increased in children under two years of age, and particularly in a child under one. Not only is the incidence increased in this age group, but mortality from incarceration likewise is most significant in the infant. If one summarizes the experience of Clatworthy,[2] Potts[7] and Thorndike[8] with such incarceration and its mortality in 3 large children's centers, the over-all mortality rate was 4 per cent in 120 cases of operative intervention for incarceration in children under 2. When one compares this with reported mortality rates of 0.6 per cent in pyloric stenosis,[1] 2.7 per cent in intussusception as far back as a decade ago[4] and practically 0 per cent in a large series of ruptured appendices,[6] this 4 per cent mortality in incarceration takes on real significance.

From the Operation

Two factors influence the thinking of those who advise parents about the correction of a hernia-hydrocele—the risk of damage to the testicle on the involved side, and the fear of recurrence.

Damage to the testicle would seem to be minimal according to two recent reports. In one[9] there was an incidence of 5 damaged reproductive organs in a series of 840 cases; atrophy occurred in 4 testicles and 1 ovary. In a smaller series of follow-ups[5] there was slight asymmetry in 4 testicles out of 239 personally followed up.

Allusion has been made to the question of recurrence, but statistical rates run well under 2 per cent in childhood herniorrhaphies, as opposed to the generally accepted rates of about 10 per cent in adult surgery for the same condition.

TREATMENT

Possible Methods

Theoretically there are five courses of action open to the physician who advises a family on hernia-hydrocele problems. First of all he may suggest doing nothing, in expectation of a spontaneous closure of the processus vaginalis. This seems to be a dangerous mode of "treatment" in view of the risk of incarceration. Although it is occasionally successful, it is rarely so in children over one year of age. A second method of handling the problem has been the use of a truss. This would seem to be of little but temporary value, except to keep the lump out of sight. It carries with it danger to the cord, especially if the truss is made of metal or hard rubber. There are occasions, in a very young child, when a yarn truss is indicated as a temporary measure until operation can be undertaken. In the third place, parents are often told that aspiration of a scrotal hydrocele will relieve the child's problem. This is poor advice when the child has a patent processus vaginalis by which intra-abdominal fluid will simply fill the scrotum again as soon as the aspiration is completed. Not only is this true, but also the danger of infection by aspiration is always present. As a fourth mode of therapy, an occasional physician will suggest injections, in an effort to obliterate the sac. This is a blind procedure which should be condemned, because it is dangerous to the cord structures and almost never effective. Finally, the time-honored and well tested surgical approach is the one which should be advocated to parents.

Prerequisites

In order to safely and properly handle pediatric hernia-hydrocele problems surgically, three things must be present. Adequate and safe anes-

thesia by an experienced person must be available; this means an anesthetist who has frequently handled small infants as well as older children. Good, efficient nursing care must be present to make the child's hospitalization as safe and emotionally atraumatic as possible. Thirdly, the surgeon undertaking children's herniorrhaphies must be one who has had wide experience in the inguinal canal of a child; he should not be one who meets a pediatric hernia only when emergency operation is mandatory.

Time of Operation

It is the firm belief of those who practice pediatric surgery extensively that a pediatric hernia-hydrocele should be corrected as soon after the time of diagnosis as is convenient, provided the child has shown evidence of being normal in other respects and has no other concomitant disease that would contraindicate the herniorrhaphy. (See also p. 995.)

Rationale

One hardly needs to elaborate upon this point at any length because of the extensive evidence adduced in the foregoing development of this subject. In summary, we believe that operation is indicated because of the high risk of incarceration (especially in the child under two), the lack of spontaneous resolution, the freedom from danger to the testicle surgically, and the low recurrence rate seen.

Method

Since the purpose of this presentation is a nontechnical one, the method of therapy can be summarized by saying that an inguinal exploration with a high ligation of the hernial sac and a reconstruction of any weakness of the internal ring constitutes the basis for adequate surgery. This is the method of treatment utilized by most pediatric surgeons throughout the country, with excellent results.

When a hydrocele is present, it is usually opened up widely in order to eliminate the fluid present. Since the hernia which has fed the fluid is corrected at the same time, this should eliminate the combination problem.

SUMMARY

The clinical evidence of an inguinal hernia-hydrocele is included within one of four manifestations or a combination of them—an inguinal bulge, a scrotal bulge, a hydrocele of the cord or a scrotal hydrocele.

The hernia-hydrocele problem is basically one in which the hope for spontaneous cure is small, and the danger of incarceration great, especially in the younger age group.

Given safe anesthesia, an experienced operator and good nursing care, surgical correction at the time of diagnosis is the therapy of choice.

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THE EMPTY SCROTUM

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When the physician is faced with the problem of an empty scrotum in his patient, a great many considerations crowd his mind. Much has been written and said on this subject, and few areas of therapeutics are surrounded by so much and so vigorous difference of opinion. From observation and from studies carried out at The Children's Hospital of Philadelphia we present a rational, physiologic approach to this problem.

DIAGNOSIS

One's first consideration must be to delineate the reason for the empty scrotum. There are five possibilities which frequently can be diagnosed by careful palpation alone: a retracted testicle, an undescended testicle lying within the inguinal canal, an absent testicle indicating either an abdominal position or agenesis, an atrophic testis, or an ectopic one.

If a testicle is felt in the inguinal region, an effort should be made gently but firmly to milk it down toward the scrotum. When a testis can be drawn to the bottom of the scrotum, even if it returns promptly to its previous location on release, it is known as a retracted or migratory testis, and no treatment of any kind is necessary. Time and normal development of the testis will increase its size and weight enough to keep it permanently in the scrotum. A structure which feels like a testicle in the canal, but cannot be moved downward or be drawn all the way to the bottom of the scrotum, must be considered a truly undescended testis. A testicle may be found lying in the abdominal wall anterior to the external oblique aponeurosis or in the perineum

From the Surgical Clinic of The Children's Hospital of Philadelphia, and the Harrison Department of Surgical Research, University of Pennsylvania Schools of Medicine, Philadelphia.

or in the medial aspect of the thigh, having descended into any of these ectopic locations. Atrophied testes may not be palpable, although the examiner frequently has a firm suspicion of this diagnosis. Failure to palpate a testis could mean atrophy, absence, or a position above the internal ring.

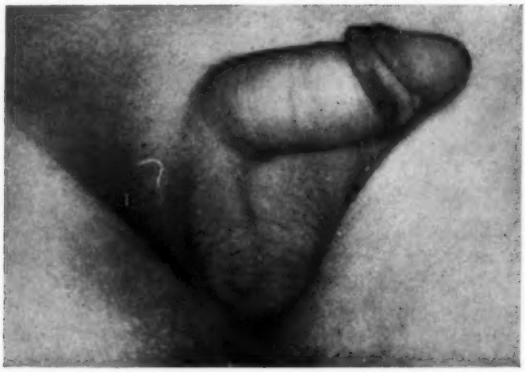

Fig. 71. Young boy with bilateral retracted testes, having nothing in either side of the scrotum on initial examination.

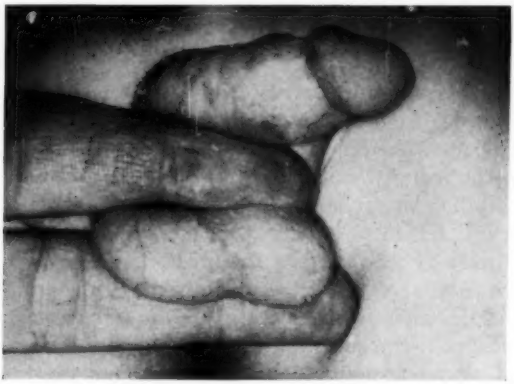

Fig. 72. After gently pushing from above and pulling from below, each testis has been brought all the way to the bottom of the scrotum, proving that these are retracted testes.

When the situation has been somewhat clarified by the physical examination, a more comprehensive plan of action may be made. In approaching this problem one must consider and cater to the concern and the often unexpressed fears of the parents of the child. A full and frank discussion of all considerations is vitally important to their

understanding and cooperation. Many mothers and particularly fathers have been given a frightening forecast of what a child's future might be by the ominous predictions of neighbors or poorly informed physicians. The prime concern of many parents is that the absence of a testis on one side will preclude a normal sexual life or reproduction. Some parents are even concerned with the possibility of homosexuality. It is our responsibility to allay these fears.

In the event of failure of both testes to descend, there is no question that some type of treatment must be instituted. We know that sterility will result if the testicles remain in the inguinal or retroperitoneal area beyond the age of puberty.

If a boy has one normally descended testis, he needs nothing more in order to develop and to function completely as a male. One might well then ask why anything should be done to a youngster who has one good testis. Before answering this question, let us first review the embryology of the testis as an approach to the whole problem.

EMBRYOLOGY

The gonad develops from the urogenital ridge in the retroperitoneal area, and there it differentiates from the kidney and begins its descent toward the scrotum. The entire process of testicular descent has been studied extensively by Backhouse (personal communication), who presents a lucid explanation of what happens. The mesenchyma of the anterior abdominal wall, in which the muscles develop, extends down into the scrotal skin. As the testis is moving downward, the processus vaginalis pushes down into the scrotal mesenchyma. Under fetal hormonal influence the mesenchyma swells with edema and with deposition of hyaluronic acid enlarging the scrotal wall. As the normal testicle approaches the scrotum, the swelling of the mesenchyma, that part just inferior to the testis being known as the gubernaculum, widens superiorly, dilating the scrotal inlet and the external inguinal ring. The enlargement is greater in a lateral direction than in a horizontal one, so that there is a relative amount of traction applied to the testis, and it comes down into the scrotum. The external inguinal ring and the scrotal inlet then fall back to a normal, snug orifice in each instance, and the processus vaginalis above the testis is usually obliterated. It would seem from some of Backhouse's observations that bands of fibrous tissue from either side or directly across the scrotal inlet are the distortions which cause incomplete descent of the testis. The undescended testicle is hence a true failure of completion of an embryologic process just as much as is an omphalocele or a cleft palate. As a matter of fact, one finds at operation the same anatomic arrangement as is found in a fetus of 20 to 24 weeks' gestational age.[7]

The exact reason for failure of descent is not known, and many assumed that the testis is basically abnormal. In any event there are several known facts which may assist our decision as to treatment. Microscopic examination of biopsies taken of undescended testes and of their normally descended mates at operation has shown that at some time between 3 and 5 years of age a distinct difference can be detected between the 2 sides.[9, 10] There appear heavier strands of connective tissue between the tubules of the testis which increase in number and thickness as time passes, gradually and relentlessly compressing the tubules and preventing the normal enlargement in tubular diameter. The cells of the testis at the same time are found to remain as a single or double layer within the tubules instead of differentiating into the usual many layers, and the nuclei do not develop in the normal pattern with the appearance of spermatogonia; however, this may not always be evident. This alteration of structure seems in large measure to be due to the increased temperature in the inguinal canal or the retroperitoneal area as opposed to the lower temperature in the scrotum.

It should be noted that even though spermatogenesis may be prevented by these changes, the androgen-producing function of the gonad is usually preserved. At the time when Leydig cells appear in the normal testis, around 10 years of age,[8] they also appear in an undescended testis and seem to carry out their usual function. They seem to be more hardy than the tubular structures.

If the undescended testicle can be placed in the scrotum, it can be permitted to develop as much potential as remains. There are those who feel that this is a vain hope and that nothing should be done, since testes so treated cannot be normally functioning ones. But our feeling is strong that gentle dissection with an appropriate operative procedure should do no harm and gives evidence of preserving function.

INDICATIONS FOR OPERATION

Perhaps the most important consideration in unilateral cryptorchidism is the psychic effect on the boy of having so grossly different a configuration from his playmates. Children do not hesitate to let others know of abnormalities they note, and a boy with one testis absent from his scrotum may be the butt of jokes, scorn or derision, which can make his life miserable. It is difficult properly to evaluate this aspect of the problem, but there is no question that many boys have become happy and extroverted after orchiopexy who were most unhappy and withdrawn beforehand. This is not to say that a good many men have not gone through life perfectly peacefully with one undescended testis, but it does merit our thought. An additional fact worth mentioning is the misinformation that frequently accompanies the teasing to which these youngsters are subjected.

Secondly, most undescended testes have an associated indirect inguinal hernia. Sometimes the hernia is symptomatic, and a certain number present with incarceration. Since the presence of a hernia is itself an indication for operation because of the unpredictability of the occurrence of incarceration or strangulation, this alone might serve as an indication for the operative approach to undescended testes.

A third fact which applies is that a true undescended testis is relatively fixed in its common position in the inguinal canal. This attachment prevents motion so that any blow to the area strikes and injures the testis, which cannot glance away as it might were it in the scrotum. A goodly number of testes so traumatized have gone on to atrophy. By operation it is possible to prevent this unfortunate chain of events.

As a fourth consideration, and a most difficult one to evaluate, we must discuss the chance of malignant transformation of an undescended testis. Dixon and Moore[5] reported a study of a large number of testicular tumors and found that the chance of malignancy developing in an undescended testicle was approximately 11 times that in a normal, scrotal testis. Their report and that of Campbell[1] would suggest that 1 abdominal testis of every 20 might be expected to undergo malignant change, and 1 inguinal testis of every 80. Carroll,[2] on the other hand, felt that malignant testicular tumors were exceedingly

rare, with an incidence of 0.0013 per cent, and that malignancy in undescended testes was even more uncommon. The objection may be raised that simply moving such a gonad into another position will not alter this propensity. But until some further proof can be brought to light, it seems wise to regard this as at least a lesser indication for operative treatment.

Finally, although one testis may be sufficient, it is nice to have a "spare."

TIMING OF OPERATION

When should one become concerned over the development of a boy with one or both sides of his scrotum empty? In the normal course of events both testes are fully descended in the newborn infant. From various reports it would seem clear that of the few which do not reach all the way at birth, a large majority will come down by the end of the first year of life. Although we feel that most of these can be differentiated by physical examination at any age, there is no urgent indication for operation until three to five years of age, as noted above, unless the associated inguinal hernia becomes symptomatic or incarcerated. This particular circumstance seems to be the only indication for which almost all surgeons, no matter how widely divergent their feelings about undescended testes and their therapy, will agree that operation with repair of the hernia and orchiopexy should be carried out.

The question of hormone treatments will come up at this time if it has not done so previously. There are many who strongly advocate the use of chorionic gonadotropin as a means of differentiating the retracted from the undescended testis, if not indeed as a method of treating undescended testes. The feeling that a good physical examination should be relied on for differentiation has led us to be leery of the use of hormones for two major reasons. First, if the anatomy of the inguinal region is such as we usually find with a hernia and a dense barrier between the fixed testis and the empty scrotum, hormones cannot correct these abnormalities. Secondly, there may be real danger in using agents with such a strong and incompletely understood potential. Recently this concern has been borne out by the revelation[3, 8] that the large doses of gonadotropin suggested in advertisements and in writings can lead to degenerative changes in the testis. The feeling in this Clinic is that the physical examination will delineate the problem and that there is no place for chorionic gonadotropin or any other hormonal injection in the treatment of this condition.

When this has been said, one is faced with the question of when to suggest operation. A fair number of authors feel that, particularly

in unilateral failure of descent, the boys should be allowed to go until they are 15 or 20 years old to see whether time will not correct the situation. This seems to be dangerous delinquency in our duty. Gross[6] and others feel it is safer to wait until the age of 9 to 11 years because the operation is safer and easier to do. Since it has been shown that the degenerative changes induced by the relative hyperthermia of the extrascrotal position begin some time between three and five years of age, we have felt that any operative procedure should be carried out ideally before that time. (See also p. 996.)

An interesting and valuable by-product of this judgment has been the finding that with practice the technical maneuvers have been easier to perform in a younger boy, and there have been fewer failures of operation. In large measure this seems to be due to an understandable anatomic occurrence. Since the testicle has not descended all the way,

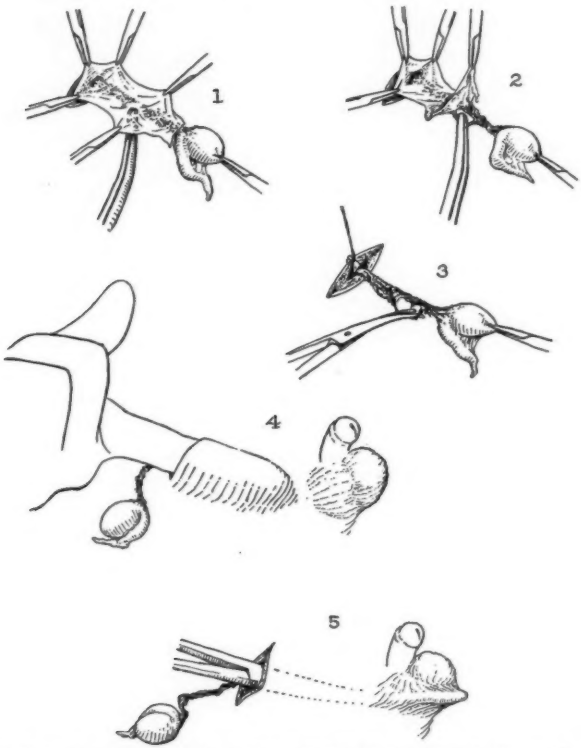

Fig. 73. Operative procedure for undescended testis. *1*, *2*, Gentle dissection of hernial sac from the cord structures, freeing it back to the peritoneal surface. *3*, The sac has been ligated, and careful dissection with removal of cremasteric fascia is carried out. *4*, *5*, Formation of a channel into the scrotum with the finger and with a Kelly clamp.

the artery finds no need for its usual length and seems to shrink. For some reason the vas deferens is almost always so long that not only does it cause no difficulty because of inadequate length, but also it is frequently tortuous and accordion-pleated. We also note thickening and binding of the vas where it is so corrugated which may well obstruct normal flow if uncorrected. The major problem in any technique of operation is the attempt to secure sufficient length of the vascular supply to permit the testis to lie without tension in the scrotum. Almost always we have little trouble in this attempt in the young patient; whereas the boy 10 years old or more commonly presents serious, if not insuperable, difficulties in this area with a poorer cosmetic result or

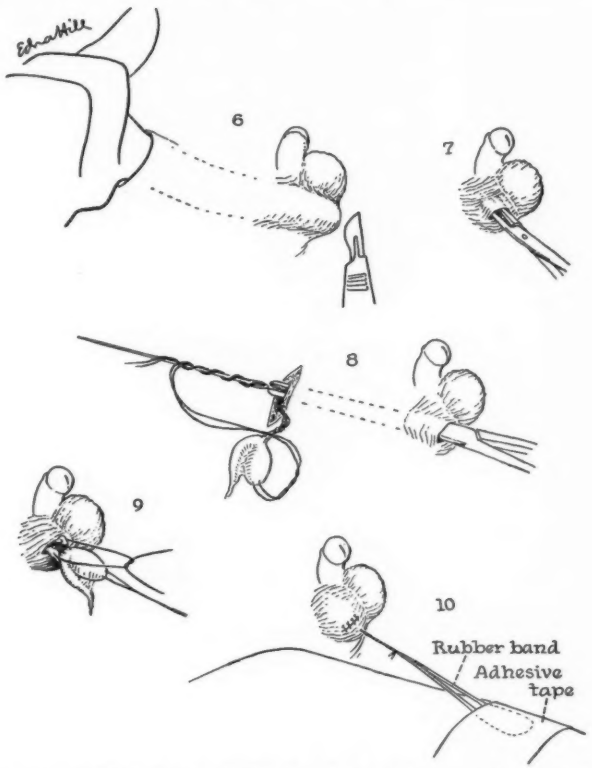

Fig. 73 (*continued*). 6, Incision in the skin of the scrotum over the tip of the finger introduced from above. 7, Creation of a subcutaneous pocket in the scrotum. 8, The silk suture through the testis is drawn down by means of a Kelly clamp introduced through the scrotum. 9, The opening in the dartos fascia is snugged about the cord structures with a suture. 10, The testis is placed in the subcutaneous pocket, passing the suture through the skin of the scrotum, and this has been secured with a rubber band and adhesive tape to the thigh.

even the necessity to carry out orchiectomy. Occasionally, when the testis is brought down as far as possible without tension at one operation, a second attempt one or two years later will be crowned with success.

TECHNIQUE OF OPERATION

The technique used in this Clinic entails operation some time before school age, usually a little earlier if the problem is one of bilateral undescended testes with at least one month between unilateral operations.

Under general anesthesia the inguinal canal is explored, and in the case of most undescended testes the gonad is found. After carefully dissecting free the hernial sac, it is transfixed at the peritoneal line, and the distal portion is excised. Gentle dissection of the cord structures is carried out then without going into the retroperitoneal area except in a very unusual case. The fine, tight bands of cremasteric fascia are teased away from vas and vessels and excised. When an appropriate length of the cord structures has been secured, often by the relatively simple maneuver of removing the hernial sac, a channel is forced into the scrotum, and a subcutaneous pocket, between skin and dartos, is created at its lowest point through a small incision in the scrotum. The testis is transfixed with a heavy silk suture which serves to draw it down into its new location and to maintain it there for approximately 1 week. The suture passes through the bottom of the scrotum and is secured to the ipsilateral thigh by means of a rubber band and adhesive tape. In placing the tape it is vital to remember that this suture is for retention of what has been achieved at operation and emphatically not for traction, which will cause spasm of the artery and atrophy of the testis. As a rule the patient is discharged home on unrestricted activity the morning after operation, and except for no baths until the suture is removed or falls out, he may do whatever he likes.

If an ectopic testis has been the indication for operation, there is seldom an associated hernia, and placement of the gonad into the scrotum is usually easily performed. It is probably wise to excise any remnants of atrophic or agenetic testicles, since they can fulfill no function. In like manner an abdominally located testis which obviously cannot be brought down by any measures or which fails to reach the scrotum after 2 operative attempts should probably be excised.

RESULTS OF OPERATION

What can be said of postoperative results of this type of approach? In the case of unilateral undescended testes no test can prove any increase in function or in the number of spermatozoa formed. Biopsy can provide only a rough estimation of the physiologic effect of operation. With a good cosmetic result, in which the surgically treated testis feels, develops and looks like its normally descended mate, we can expect that at the very least we have repaired a hernia and eliminated a major source of emotional concern at an age when conformity to the norm is of much importance. When both testes have failed to descend, proved fatherhood following orchiopexy, as found by Gross and Jewett[6] in 79 per cent of 38 boys, bears out our feeling that the operation is successful.

The many biopsies which Charny[3, 4] has done over the years have

helped expand our understanding of the problem immeasurably. He has been led by his studies to feel that most testes brought down by operation are basically so abnormal or have been so damaged by the procedure that they cannot be spermatogenic. Most of the testicles he has examined by this method were operated upon by surgeons other than himself, using a wide variety of surgical techniques, many by means of the Torek operation. We know that any damage to the spermatic artery will lead to testicular atrophy, and the fixed tension on the artery caused by the Torek maneuver of suturing the testis to the thigh leads to arterial spasm. For this reason the Torek procedure has advisedly been abandoned by most pediatric surgeons, and some such technique as that described above has become the method of choice for treating undescended testes. It is hoped that a protracted follow-up with biopsies of the gonad will bear out our strong feeling that this type of treatment not only does no harm to the testicle, but also allows it to progress to normal development and function.

SUMMARY

The problem posed by an empty scrotum can be clarified frequently by a careful physical examination, which should attempt to differentiate the undescended testis, the retracted testis, the ectopic testis or the atrophic or agenetic testis.

The retracted testis requires no treatment to assure normal position and function.

There should be a full and frank discussion with the parents and with the older boy to apprise them of the possibilities.

The reasons for and timing of operative therapy are fully discussed.

The use of chorionic gonadotropin should be discouraged.

Operation should be performed before school age, if possible, with removal of the hernial sac and placement of the testis in a subcutaneous scrotal pouch. In the case of bilateral failure to descend, early operation is advised in order to preserve as much function as possible.

A technique of surgical approach is described which leads to good functional and cosmetic results without the dangers inherent in an operation which utilizes fixed traction on the testis.

Boys presenting this problem deserve careful thought and judgment in order to preserve as much normal function as possible and to guard against emotional trauma.

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CONSTIPATION AND MEGACOLON

Diagnosis and Pitfalls

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Constipation is one of the frequent complaints encountered in medical practice and often may be traced to habit patterns developed in early childhood. It is indeed fortunate that medical science has dispelled the idea that failure to have a daily colonic evacuation may be harmful to the health through absorption of toxins from the intestinal tract. Whereas formerly a child would be given strong purgatives to relieve constipation, we now know that large collections of fecal material in the colon exert only a local mechanical effect. Fecal masses in the colon do not impair growth and development of the child, and regular use of potent laxatives may cause a mucous colitis or chronic electrolyte depletion. Therapy in recent years has been directed at the cause of the constipation rather than at the symptom itself.

HABIT CONSTIPATION

The majority of children with constipation fall into the group that has been labeled habit constipation. With this syndrome the symptoms are ordinarily not manifest until after toilet training has begun or when the child is two or three years old. Child psychiatrists have pointed out that mother-child antagonism and animosity build up emotional tensions which manifest themselves in the child as constipation. He holds back the fecal material and ignores the local sensations of the urge to defecate. As the fecal material accumulates, it becomes dehydrated and inspissated, causing painful abrasions and fissures in the anal canal during infrequent evacuations; thus a local mechanism augments the child's habit of holding back. With time the rectum and colon dilate and become flaccid, and peristaltic activity becomes ineffectual.

The parents may not seek medical aid until the final symptom of this

chronic constipation syndrome has appeared, that of fecal staining. This is the chief complaint of school children who have had the syndrome for several years and have developed large inspissated masses of feces in the lower colon. The relatively soft material in the upper colon is pushed down around these fecal masses and is extruded involuntarily through the anus much the same as happens to a patient with diarrhea. The fecal staining may occur diurnally or only at night when the child is asleep. Another problem in the school child with this syndrome is evacuation of such large fecal masses that the sanitary facilities of the school may be obstructed. This again prompts the child to hold back evacuations. Thus the complaints of the child with constipation are related to social problems, such as soiling and massive evacuations, rather than to the child's health. The colon and abdomen are able to accommodate large collections of material causing little discomfort to the child or distention of the abdomen. If the colon fills the pelvis and presses on the urinary bladder, frequency, urgency and even urinary incontinence may occur.

Upon examination the child with constipation appears in good health; growth, development and nutrition are normal. Collections of gas are not ordinarily found in the colon, since these children are able to pass flatus without difficulty. A fecal bolus is palpable in the lower abdomen, and on rectal examination the mass of feces may be easily identified, beginning just within the anal sphincter.

In the majority of children with habit constipation the diagnosis may be made on the basis of the history and physical examination. Constipation beginning at the age of one or two years, and not accompanied by abdominal distention, is very likely on a habit basis. Roentgenographic examination is rarely necessary; but in cases that have some of the features of congenital megacolon, a barium enema is helpful to rule out this condition. In the cases of habit constipation the colon is dilated from the anus proximally for varying distances, depending on the amount of fecal material collected (Figs. 74, 75). The barium mixture is evacuated easily, although the inspissated fecal material may be retained.

Successful management in the case of persistent, severe constipation requires patience in the careful investigation and adjustment of the parent-child relationship. If available, this program may be best carried out by a medical social worker and psychiatrist through a habit clinic. It is important to start definite treatment before fecal soiling begins, for the combination of symptoms is extremely resistant to therapy. A child with fecal soiling should be admitted to hospital and the inspissated feces extracted under anesthesia if necessary. It is surprising how often they will have daily colonic evacuations while in the hospital, but will return to the habit constipation pattern as soon as they are back in the home environment.

Fig. 74. Roentgenogram of a child with severe chronic constipation. The rectum and sigmoid colon are dilated and filled with a fecal mass.

Fig. 75. Spot roentgenogram taken during filling of the rectum for the same child as in Figure 74. The dilatation of the colon extends to the anal canal.

RECTAL BIOPSY

In certain cases of severe constipation when the x-ray study does not completely rule out Hirschsprung's disease, we have found the rectal biopsy a useful aid in diagnosis.[1] The biopsy is easily obtained and gives definite, reliable information about the diagnosis. At the time of the fecal disimpaction under anesthesia, a small bit of the rectal wall may be obtained 2 or 3 cm. above the anus. On study of the histologic sections the presence of ganglion cells in Auerbach's plexus rules out the diagnosis of Hirschsprung's disease. The incision in the muscular coats is closed with interrupted silk and in the mucosa with interrupted catgut sutures. The child may be discharged from the hospital the following day with no particular postoperative precautions.

During 5 years' experience with this diagnostic procedure more than 100 biopsies have been performed. One fatality has been encountered in a newborn with Hirschsprung's disease who died at the end of the procedure with a cardiac arrest. Postoperative complications have been few, there being no perirectal abscesses or fistulas. One child early in the series required additional sutures because of oozing from the incision. In this group of patients the majority proved to have chronic constipation.

DIAGNOSIS OF HIRSCHSPRUNG'S DISEASE

The condition usually confused with habit constipation is congenital megacolon, or Hirschsprung's disease. The child with Hirschsprung's disease has symptoms of intestinal obstruction within the first few days of life, having difficulty passing meconium. The constipation continues to give symptoms intermittently throughout infancy and childhood, and bouts of abdominal distention are a prominent feature. Soiling and the passage of large fecal masses are never a problem with these children. Their chief complaint is related to the impaired health caused by the megacolon. On examination these children have a large abdomen filled with the colon containing both gas and fecal material. Feces usually are not palpable in the rectum, since the aganglionic segment is empty of feces. A barium enema will indicate the diagnosis by demonstrating a narrow aganglionic segment in the rectosigmoid area and the dilated left colon containing fecal material (Fig. 76). Rectal biopsy is rarely necessary to make the diagnosis in the older child. In the neonate, however, biopsy is routinely necessary to rule out other conditions causing a similar picture. (See also p. 1000.)

IMPERFORATE ANUS

The child with imperforate anus has persistent difficulty with colonic evacuation resulting in constipation. The postoperative care of the infant after anoplasty does not require daily enemas because the soft

Fig. 76. Roentgenogram of a child with Hirschsprung's disease. The left colon is widely dilated, but the rectosigmoid is narrow. Peristaltic waves were seen in the left colon during fluoroscopy examination, but none was identified in the rectosigmoid area.

stools of infancy can be easily evacuated. As the child becomes older, however, particularly at the time of toilet training, daily enemas should be started. Without enemas the child after an anoplasty evacuates incompletely and tends to collect large amounts of fecal material. The rectum may become dilated and resemble a megacolon. To rehabilitate these children it may be necessary to revise the anoplasty to obtain a large enough opening so that the finger can be inserted easily. A regimen of daily enemas should be begun after the child has been disimpacted. We have never seen Hirschsprung's disease associated with this anomaly.

Some children with constipation have a tight opening and on careful study have a mild form of imperforate anus. The anal canal is incompletely formed and represents essentially a fistula from the rectum to the perineum. If anal dilatation and daily enemas do not relieve the child of the constipation symptoms, then an anoplasty may be necessary to obtain an adequate opening.

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RECTAL BLEEDING IN INFANTS AND CHILDREN

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Bleeding per rectum is a common symptom in the pediatric age group. It may be of little significance, or the child's life may depend on prompt detection and correction of its cause. The decision into which of these categories a case falls usually presents no great problem. There are, however, a number of cases in which the cause of the bleeding is obscure and presents a real challenge to the most experienced physician.

The first step in the approach to this problem is to confirm the presence of blood in the stool by a *guaiac test*. On occasions stools colored by foods (beets, tomatoes, chocolate, and so on) are indistinguishable by inspection from those containing blood. A significant number of riddles can be solved immediately by this simple test.

A problem which occurs occasionally in the newborn is the differentiation of gastrointestinal bleeding from blood ingested during birth. This can be readily accomplished by placing some of the blood in 10 cc. of distilled water, then adding 0.2 cc. of 10 per cent sodium hydroxide. If the solution turns brown, adult hemoglobin is present. If it remains pink, fetal hemoglobin is present (*Apt test*[1]).

Children are much more likely to swallow blood from the nose or pharynx than adults. As a result, a child may pass a tarry stool a day or two following a nosebleed.

It is incorrect to refer to all rectal bleeding as a melena. *Melena* is the passage of tarry stools, indicating that the blood is from high in the gastrointestinal tract. Melena is usually associated with hemateme-

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sis. The passage of bright red blood per rectum is not melena, but is *hematochezia*. Lesions producing hematochezia occur in the lower small bowel, colon or anorectum.

Bleeding in the gastrointestinal tract may be the result of a generalized bleeding disorder or of some local lesion in the alimentary canal. Generalized diseases which may produce intestinal bleeding in the pediatric age group include hemorrhagic disease of the newborn, anaphylactoid purpura, thrombocytopenic purpura, scurvy, leukemia and other blood dyscrasias. None of these conditions produces only gastrointestinal bleeding; a definite search should therefore be made for other evidences of bleeding elsewhere in the body—hematuria, petechiae, ecchymoses, and so forth.

ETIOLOGIC CONSIDERATIONS

Hemorrhagic disease of the newborn occurs only in infants up to one week of age who have not received vitamin K and is due to a deficiency in prothrombin associated with a deficiency of serum prothrombin conversion accelerator (Factor 7) and plasma thromboplastin component (PTC). It can be corrected by giving as little as 2.5 mg. of vitamin K_1 intramuscularly or intravenously. Any bleeding due to this deficiency will stop within four hours when adequately treated.[3] A word of caution should be injected about overdosage of vitamin K, which has on occasion produced jaundice in infants.[2]

Anaphylactoid purpura producing gastrointestinal bleeding is usually associated with abdominal pain and purpuric spots, particularly on the lower extremities. There is frequently an antecedent history of a respiratory infection. One particularly alarming feature of this disease is that on rare occasions bleeding into the wall of the bowel may produce a mass which acts as a lead point for an intussusception. The symptoms due to this complication are most difficult to distinguish from those produced by the primary disease. A barium enema is of no help, since these are usually small bowel intussusceptions. Plain x-ray films of the abdomen may show dilated loops of small bowel. If the pain is due to the purpura, primarily, it should subside with 24 to 48 hours of steroid therapy. If the pain is due to the secondary intussusception, these steroids will have little effect on the discomfort. (See also p. 1082.)

Thrombocytopenic purpura is easier to recognize because of the abnormal platelet count associated with an otherwise essentially normal peripheral smear. This is easily detected by examination of the routine blood smear and noting an absence or a paucity of platelets.

Scurvy may cause rectal bleeding, but is almost always associated with changes in the gums and long bones in an irritable child. There should also be a history of deficient vitamin C intake.

Leukemia may produce rectal bleeding, but this usually occurs late in the disease when the diagnosis is reasonably obvious as the patient is examined and the smear scrutinized. Other blood dyscrasias may on rare occasions produce gastrointestinal bleeding.

Rectal bleeding which occurs from a local lesion high in the alimentary canal (esophagus, stomach or duodenum) is usually associated with vomiting of blood. The stool may be grossly normal, containing only occult blood. These lesions may produce melena or tarry stools, or, if the bleeding is brisk, the stool may contain bright red blood.

Bleeding *esophageal varices* are the most common cause of massive upper gastrointestinal bleeding in the pediatric age group. They produce sudden massive hemorrhage or may produce repeated loss of small quantities of blood. The spleen is usually palpable, since the varices are the result of portal venous bed block. Bromsulphalein and other liver function tests which are frequently helpful in implicating varices as the cause of acute gastrointestinal bleeding in the adult are of little help when dealing with children. Half of the children with varices have an extrahepatic portal vein block and normal livers. The most useful diagnostic study is a barium swallow, which will usually demonstrate varices. If the bleeding is massive, a Sengstaken-Blakemore tube may be a diagnostic as well as a therapeutic instrument. In children too small for these tubes a Miller-Abbott tube with a new balloon can be used. The tube is passed into the stomach and the balloon inflated with 75 to 100 cc. of air. The balloon is then brought up against the cardia of the stomach, compressing the varices as they pass from the stomach into the esophagus.

Treatment of an acute episode of bleeding varices consists in adequate blood replacement, sedation and the use of a ballooned tube. If the bleeding is not controlled by the balloon, the varices must be attacked directly by transthoracic ligation. Once the acute episode has been controlled, more definitive treatment must be undertaken. The best results are obtained from a direct venous anastomosis between a portal bed vein and a systemic vein. The splenorenal shunt has given the most satisfactory results in children. A portacaval anastomosis is not possible in the majority of children because of portal vein block. In those patients who have had a splenectomy with or without splenorenal shunt and are bleeding again, an interposition of colon or an esophagogastrectomy is the procedure of choice. All patients operated on for esophageal varices should have a percutaneous splenoportogram prior to the shunt procedure to determine accurately the location of the block and the status of the portal vein. (See also p. 999.)

A *hiatal hernia* may cause blood to be passed per rectum, owing to reflux peptic esophagitis and bleeding. These patients usually vomit frequently and have substernal pain when lying flat. The bleeding is

rarely massive, but may account for a considerable blood loss over a period of time. The diagnosis is made by barium swallow, and on occasion esophagoscopy is helpful in evaluating the esophagitis. (See also pp. 946, 1038.)

A number of these children will respond to treatment with frequent small feedings, antacids and avoidance of the recumbent position. If this does not cause a relief of symptoms, the hiatus hernia should be repaired.

Duodenal or *gastric ulcer* is a rare cause of upper gastrointestinal bleeding in children. When ulcers do occur, they are frequently of the Cushing or Curling type. Any child with a bleeding ulcer should have an adequate central nervous system evaluation. The diagnosis is usually made with difficulty by barium study of the stomach and duodenum. Since these ulcers are usually acute with a shallow crater, they may be difficult to demonstrate by x-ray.

Treatment consists of frequent feedings, antacids, atropine derivatives and blood replacement. If this regimen does not control the bleeding, a laparotomy should be carried out. The ulcer should be exposed and the bleeding point ligated. A subtotal gastric resection or other more radical procedure has no place in the treatment of this lesion in children. The children who have been treated by simple ligation of the bleeding point and placed on an ulcer regimen have done well without recurrence of bleeding or ulcer. They have none of the nutritional problems which are apt to occur in a growing child who has been subjected to a more radical procedure.

Other conditions causing bleeding from the upper gastrointestinal tract are uncommon. Occasionally *gastritis* with repeated vomiting may cause bleeding. *Neoplasms* in this area in children are almost nonexistent. *Duplications, hemangiomas* and *ectopic pancreatic tissue* do occur and cause bleeding, but again this is most uncommon.

Lesions in the small bowel usually produce only rectal bleeding, but on rare occasions hematemesis is produced if the lesion is high in the jejunum or if the bleeding is massive.

Meckel's diverticulum (see also p. 1091) is the most common lesion of the small bowel producing gastrointestinal bleeding. The bleeding is from ulceration in the adjacent ileum secondary to the secretions from the ectopic gastric mucosa or pancreatic tissue occurring in the diverticulum. The bleeding is usually intermittent and of fair magnitude. It may be preceded by a day or two of vague abdominal discomfort, but usually there is no pain. The stools are at first dark red, then become bright red as the bleeding continues. The bleeding usually subsides after a varying period of time, though an exsanguinating hemorrhage may occur. The bleeding usually recurs a few days to several months later.

Roentgen studies are of no value in the diagnosis of Meckel's diverticulum. The decision to operate must be made on clinical grounds alone. The treatment is surgical removal of the diverticulum. In most instances this can be accomplished by a simple excision. An inflamed or a perforated ulcer at the base of the diverticulum occurs on rare occasions, necessitating resection of the ileum with an end-to-end anastomosis.

Duplication of the intestinal tract may cause bleeding in one or two ways. The duplication containing gastric mucosa may communicate with the intestinal tract, its acid pepsin secretion producing ulceration. The duplication which does not communicate with the gut, lying, as it does, often within the leaves of the mesentery, may press on the mesenteric vessels, causing necrosis, slough and bleeding of the adjacent intestine. Bleeding in the first type may be massive or small in amount, while in the second type is seldom extensive. The latter type, however, is likely to be associated with an abdominal mass.

Roentgenographic examination generally gives helpful diagnostic information, but on occasion may fail to reveal a lesion. Treatment consists in excision of the duplication along with this adjacent intestine, since most of these lesions have a muscularis which is common to it and the adjacent gut. Intestinal continuity is re-established by an end-to-end anastomosis or, in the very sick child, by a Mikulicz resection.

Intussusception may under unusual circumstances have bleeding as its primary symptom. This bleeding is usually of the currant jelly type. More often cramping abdominal pain is a prominent feature which is usually associated with vomiting and an abdominal mass. More often than not the child previously has been well and is between 6 and 24 months of age.

A barium enema is diagnostic in the common ileocolic type of intussusception, but is of no value in the ileo-ileal variety. Treatment is by surgical reduction of the intussusception in most instances. There are a few cases in which reduction cannot be accomplished. In this situation the safest form of resection, in our experience, is a Mikulicz exteriorization procedure followed five to seven days later by extraperitoneal closure of the ileostomy stoma.

Small bowel polyps are uncommon. Their most frequent symptom is rectal bleeding. The bleeding may be occult or may be dark red blood mixed with stool. It is not massive. Small bowel polyps are difficult to demonstrate roentgenographically. Jeghers et al.[6] have pointed out the diagnostic importance of melanin spots in the perioral skin and mucous membranes in patients with intestinal polyps. Treatment is removal of the polyp or polyps if it be single or scattered, or resection of the bowel if multiple polyps are confined to a small segment. The patient with extensive polyposis of the small or small and large bowels presents a real problem in management if the bleeding is of any quantity.

Hemangiomas of the intestinal tract are rare entities and, when they do occur, are usually associated with cutaneous hemangiomas. *Hereditary hemorrhagic telangiectasia* (Osler-Weber-Rendu syndrome) may involve the gastrointestinal tract. The gastrointestinal bleeding is usually of small quantity, and the treatment nonoperative supportive measures. If the bleeding is massive, resection of the involved portion of the gut will be necessary.

Midgut volvulus (see also p. 969) is a rare cause of rectal bleeding which is peculiar to the pediatric age group. It occurs in patients with malrotation whose midgut twists (usually clockwise) around the narrow mesenteric attachment compressing the superior mesenteric vessels. The volvulus also produces an intestinal obstruction which is apparent on x-ray, but this may appear later as the twist tightens. Thus bleeding per rectum may be the first symptom. Treatment consists in reduction of the volvulus and division of the lateral peritoneal bands crossing the duodenum to the transverse colon (Ladd's procedure).

Bleeding from local lesions in the colon produces hematochezia and red blood in the stool; it is never associated with hematemesis, and the stools are not tarry.

Polyps (see also p. 998) occur much more commonly in the large than in the small bowel. Their most common symptom is bleeding. When the polyp is large and is located low in the rectum, some tenesmus may be present. A rare low polyp is passed out of the anus. Colonic polyps may be single, scattered or multiple. If a polyp is known to be present in the rectum, a barium enema should be done to determine the presence or absence of other colonic polyps. In children it is better to do a barium study before sigmoidoscopy, since a general anesthetic is usually needed for this latter procedure. If a second polyp is present above the reach of the sigmoidoscope, laparotomy can be undertaken during the same anesthesia.

Single or scattered polyps of the rectum or rectosigmoid are snared and their stalk fulgurated through a sigmoidoscope. Those located at a higher level are approached by a laparotomy and removed through a colotomy. Colonic resection advocated by some for colonic polyps in adults is unwarranted in children and should not be done for single polyps.

Polyposis of the colon, familial or nonfamilial, may produce rather pronounced blood loss. The multiple polyps usually involve the entire colon, though occasionally they may be segmental. Carcinoma developing in these children after puberty is a definite possibility. (We have never seen a carcinoma develop in a polyp before puberty.) In the growing child every effort practical should be made to maintain normal bowel movements. In view of this, a subtotal colectomy with ileoproctotomy is performed for polyposis of the entire colon. The rectum distal

to the anastomosis is visualized and all remaining polyps biopsied and fulgurated every three to six months. If at any time changes suggestive of malignant degeneration occur, the rectum is removed and a permanent ileostomy performed.

Chronic ulcerative colitis is a fairly common cause of rectal bleeding during the prepuberal and adolescent years. The stools are usually loose, occurring several times a day. Symptoms are apt to have been present for some time before medical aid is sought. If the disease has been present for any period of time, there is usually a weight loss or a failure to gain weight. Fever may be a prominent symptom, particularly during exacerbation of the disease. Diagnosis is by barium enema which shows loss of haustrations, shortening and fixations, ulceration and pseudopolyp formation. Sigmoidoscopy may show definite ulceration or only a red, inflamed friable mucosa, which bleeds when touched.

Treatment is medical with low residue bland diet, nonabsorbable sulfonamides, vitamin, iron, and transfusions as needed. Steroids are frequently of value. In spite of these measures there are some patients whose symptoms do not abate, and there is progression of the disease. In some of these a subtotal or total colectomy may be lifesaving. In others it will produce a striking improvement in their general health and a prompt weight gain.

Infectious bacillary colitis produces a bloody diarrhea accompanied by a high fever and acute dehydration. Diagnosis is confirmed by culturing Salmonella or Shigella from the stool. Treatment is by appropriate antibiotics.

Rectal prolapse is usually associated with the passage of some blood per rectum. Prolapse can usually be managed successfully in children by a nonconstipating diet and strapping the buttocks together. It is helpful for the child to have his bowel movements in the recumbent position on a bedpan rather than in the sitting position. Rarely is an operation necessary.

Fissure in ano is without doubt the most common cause of rectal bleeding in the pediatric age group. The child is constipated and has pain on defecation, and the blood is streaked on the outside of the stool. The bleeding from a fissure is never massive, and if there is a significant fall in the hematocrit value, some other cause than an anal fissure must be sought to explain the gastrointestinal bleeding. The fissure can usually be seen simply by spreading the buttocks. If no fissure is seen, the lubricated conical tip of a centrifuge tube should be introduced into the anus. By peering down the lumen of the tube, fissures inside the anal margin are readily seen.

Treatment of anal fissure in children is nonoperative. The first important step towards curing this malady is a good stretch of the anal sphincter. This should be sufficient to paralyze the sphincter for several days. This will relieve the spasm of the sphincter and much of

the pain. The stools should be kept soft. In the infant this is easily accomplished by adding more carbohydrates to the formula. In older children a mild laxative will accomplish this purpose. Mineral oil should not be used for this over a long period of time because of interference with absorption of fat-soluble vitamins and because of the possibility of lipoid pneumonia. Sitz baths prior to bowel movements are helpful in healing the fissure and relieving the discomfort.

MANAGEMENT

Presented with a patient with rectal bleeding, it is worth while to approach the problem in a systematic fashion. If a patient has a good history for a fissure, if a fissure is demonstrated and there is no significant drop in the hemoglobin, the fissure should be treated and the child not subjected to elaborate diagnostic studies. Fully half of all children with rectal bleeding will be cured by this simple approach.

In the other half of children, the bleeding is due to a more serious and subtle lesion. A good detailed history about excessive bleeding and bruising, bleeding disease in other members of the family, and past history of hemarthrosis or of extensive soft tissue bruises is essential to rule out congenital bleeding disorders. A thorough search for other evidence of bleeding disorders—petechiae, purpuric spots, bleeding gums, enlarged spleen, and so forth—must be made. If the prothrombin time is normal, and if the peripheral smear contains adequate platelets and no abnormal cells, no further hematologic studies need be undertaken. Bleeding and coagulation times as usually done clinically are so inaccurate that they are of little value. If history, physical examination or simple blood studies show any evidence of a generalized bleeding disorder, the patient should be evaluated by one competent to carry out a more detailed hematologic investigation.

If melena is present or if there is associated hematemesis, the upper gastrointestinal roentgenographic examination is more apt to be rewarding. If the blood is bright red, barium enema is more likely to demonstrate the source of bleeding. The barium enema should be followed by sigmoidoscopy. In a good number of cases the diagnosis will be made by these studies, and then the condition can be appropriately treated.

Unfortunately, there is still a disappointingly large number of patients in whom a definitive diagnosis cannot be made. This group of patients must be considered candidates for diagnostic laparotomy. If a patient has had one documented massive hemorrhage requiring transfusion, exploration should be done. If he has never had a massive hemorrhage, but has continued to run occult blood in the stool and to drop his hemoglobin sufficiently to require transfusion on two or more occasions, a laparotomy probably should be done. When a

laparotomy is done under such circumstances, the surgeon must be willing to make a careful exploration of the entire intra-abdominal gastrointestinal tract for the source of bleeding. The cause of the bleeding will be found and corrected in only about half of this group. The parents must, of course, be aware of this before laparotomy. The only encouraging feature of this is that the lesion which is apt to produce exsanguinating hemorrhage (Meckel's diverticulum or communicating duplication with gastric mucosa, and the like) is usually readily apparent and can be relatively easily removed. In the face of the disappointing news that the cause of the bleeding has not been corrected at laparotomy, the parents and physician derive some consolation from the fact that the child probably does not have a lesion which will allow him to bleed to death at home some night. In any large series of patients with rectal bleeding there will be a few for whom in spite of a complete diagnostic evaluation, including a laparotomy, there will be no answer as to the cause of the bleeding and who will continue to bleed. The management of these patients and their families will tax the ingenuity of even the most ingenious.

A word should be said about one special group of patients, that is, the newborn infant with massive gastrointestinal bleeding. It is usually difficult or impossible to find the source of bleeding in these infants. Some of them no doubt bleed as a result of gastric or duodenal ulceration. Some bleeding is due to hemorrhagic disease of the newborn. These patients should be managed conservatively, given 2.5 mg. of vitamin K_1 intramuscularly or intravenously, and given adequate blood replacement. Laparotomy is undertaken at a great risk with little chance of finding a surgically correctable lesion.

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EARLY EVALUATION OF MUSCULOSKELETAL LESIONS BY THE PEDIATRICIAN

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Early evaluation of musculoskeletal lesions by the pediatrician avoids increasing deformity with growth and enables early training in the use of the extremities.

FOOT

The feet, though subject to less and less function with increasing mechanization of our lives, are continuing as objects in infancy to be criticized by relatives, in childhood to be exploited by shoe salesmen, in middle age to affect one's disposition, and in old age to be accepted for punishment.

In the infant, recognition of foot deformity is generally not difficult. Some overemphasis has been placed on early treatment. This was probably done in the past in order to prevent neglect through postponement of obtaining a consultation with an orthopedic surgeon. Normally we expect the foot of an infant to dorsiflex to a right angle. When the foot is held in dorsiflexion, the midline of the patella is in line with the crease between the first and second toes.

A frequent deviation from this is talipes equinovarus, or *clubfoot*. Here the foot remains in equinus, the sole of the foot is turned towards the opposite foot, and the toes are toward the midline of the body (Fig. 77). There is an apparent rounding of the lateral border of the foot, and the heel is tilted inward. The latter is obvious when the child is turned over on his abdomen. The treatment of clubfoot should be

Fig. 77. Clubfoot (talipes equinovarus) shows adduction of the fore part of the foot and varus of the heels.

started within the first weeks after birth. The most successful management of the clubfoot is by the Hoke method, in which plaster of paris casts are repeatedly applied to gradually correct the adduction, later the varus, and finally the equinus position of the foot. The correction is attained through frequent changes of the casts, in the young infant as often as once a week and in older infants at two-week intervals. In older children the casts may be wedged by cutting a pie-shaped piece out of the cast on the side toward which the correction should take place. It is not possible to specify the length of time required for correction of clubfoot, since rigidity of the foot and the association of other deformities delay the progress. Rarely is it necessary to do open surgery for correction of a clubfoot. Division of joint ligaments (capsulotomies), anterior tibial tendon transplant, "heel cord" lengthening are reserved for older children in whom there has been a recurrence of deformity. Recurrences of deformity after initial correction can be prevented by the use of special shoes and night splints for an indefinite number of months or years.

Pes adductus (adduction of the forepart of the foot) resembles clubfoot, but does not have any deformity or restriction of motion of the heel. It can, however, result in a severe flat foot when the child attains adolescence. The toes of the adducted foot turn inward. A wrinkle is apparent transversely across the arch of the foot. The foot does not have a contracted heel cord, but the forepart, when pushed outward, will fall right back into the adducted position. X-ray studies show the foot (metatarsal bones and phalanges) to be angled 30 degrees or more toward the midline at the tarsal metatarsal joints. The adducted foot can be readily corrected with repeated plaster casts to hold the meta-

tarsals in abduction. This foot is much less resistant to correction than the clubfoot. Changes of plaster casts over a period of two to four months result in correction. Precaution, however, must be taken after satisfactory correction has been obtained. The foot must be held in the corrected position. This can be done by the use of splints, the most popular type of which is the Dennis-Brown splint, which consists of a bar placed between two shoes, either of which can be adjusted on a ratchet permitting outward rotation. During the day, and for weight bearing, a special "out-flared" shoe is used. This shoe is built to hold the foot away from the midline.

Fig. 78. Metatarsus primus varus in a girl of 5 years. Note the shorter first metatarsal and its separation from the second metatarsal.

Often accompanying pes adductus is *metatarsus primus varus* (Fig. 78). This is adduction of the great toe. The great toe will spread from the other toes toward the midline. This has been called the aboriginal foot, the great toe simulating a thumb. This position of the great toe, of course, was useful when our prehistoric antecedents were adept at climbing trees. The structure of this foot is such that difficulties are prone to occur with increasing weight and particularly in the dress shoes that women accept with as much resignation as childbirth. The adducted first metatarsal is shorter than the other metatarsals. This upsets the so-called tripod arrangement of the arches of the foot. As described by Dudley Morton, the weight-bearing legs of the tripod are the tubercle of the os calcis, the fifth metatarsal head and the first metatarsal head. The first metatarsal head, being shorter and more toward the midline, permits pronation of the foot (flatfoot position) and transfers the weight to the second metatarsal head. The individual, therefore, puts strain on the longitudinal and anterior arches of the

foot. It is necessary for children to wear a longitudinal arch pad with a flange extended beneath the first metatarsal head in order to bring the weight where it should normally occur. In young ladies all conservative measures are abandoned for conventional high-heel torture. Here we have the weight thrust upon the metatarsals. The foot is held by wedging the toes in a small cornucopia at the front of the shoe. This results in the toes curling in a "hammer" position, which is aided by the anterior arch strain and resultant spasm of toe muscles. The great toe is forced against the other toes as directed by the tip of the shoe. This results in a hallux valgus deformity and subsequent development of a bunion.

At times we are confronted with a *calcaneus foot*. As we look down upon the recumbent infant, the top of the foot is folded against the lower leg, and the sole of the foot is apparent (Fig. 79). There is generally an associated valgus deformity in which the foot is rolled laterally. In the calcaneus foot, which is the opposite of a clubfoot, the anterior tibial, the peroneus tertius tendon, and the toe extensor tendons are shortened; the plantar flexors, heel cord, toe flexors, and so on, are stretched out. The correction of this foot is again by plaster casts changed at weekly intervals in order to obtain more plantar flexion and a varus position (toes pointing downward and inward toward the midline of the foot). Although correction takes place within a few weeks, it is difficult to maintain it, since the plantar flexors of the foot for some weeks do not take up the slack in their tendons. A modified Dennis-Brown splint or plaster of paris splint to hold the correction is necessary for some months. If the calcaneovalgus foot continues unrecognized until the child begins to walk, it is necessary to use built-up shoes with a 3/16-inch leather wedge on the inner border of the sole

Fig. 79. Calcaneus foot on the left, clubfoot on the right.

and the heel, and increase the elevation of the heel to force the forepart of the foot into more equinus. These measures correct the weight thrust on the foot and permit the heel cord to shorten.

The *plantar-flexed talus* is another foot deformity of early childhood which may not be recognized until the child is weight bearing (Fig. 80). In this foot the talus (astragalus) has taken a vertical position, and the head of the talus points toward the plantar surface. The navicular articulates with the dorsal lateral surface of the head of the talus. This deformity can be attained in a clubfoot if the foot is forced into dorsiflexion before varus position of the heel is corrected. In either case, whether due to too rapid correction or to a primary position, the foot must be brought back into equinus and adduction and

Fig. 80. The plantar flexed talus (astragalus) is in a vertical position.

the equinovarus of the hind part corrected. In the foot with a primary talus deformity a high leather counter shaped over a plaster mold of the foot to extend above the medial malleolus is fitted in a high-top shoe with a rigid shank until a better position of the foot is attained. For older children it is necessary to utilize a metallic or plastic arch support. In resistant cases heel cord lengthening, a posterior capsulotomy of the foot, and occasionally a transfer of the anterior tibial tendon to the neck of the astragalus are relied upon for correction. All corrective measures are accompanied by exercises to stretch the "heel cord" and strengthen the extensors (dorsiflexors) of the foot and short flexors of the toes.

Another condition causing a flat foot is that of an *accessory navicular bone* (Fig. 81). There is either a second epiphysis or a lack of

union of the tubercle of the navicular. This results in downward projection of the navicular in the longitudinal arch of the foot. A heavy callus forms over the prominence. Kidner stressed that this deformity affected the supportive action of the posterior tibial tendon, which normally is attained by the tendon passing beneath the navicular bone to insert in the cuneiforms and metatarsals. Such a foot must have support in childhood by means of a longitudinal arch pad and plastic or metallic arch plates in older years, accompanied by muscle exer-

Fig. 81. Note the prominence on the inner side of the foot due to the accessary navicular bone.

cises to improve the balance of the foot. Surgical correction is achieved by removing the accessory bone, transplanting the anterior tibial tendon beneath the navicular, and stabilizing the scaphocuneiform joint with a bone graft (Hoke procedure).

Flatfoot is frequently associated with *peroneal spasm*. As one looks at the lateral side of the flat foot, the peroneal muscles bridge or stand out as a ridge behind and beneath the lower fibula or lateral malleolus. This foot cannot be pulled into corrected position. The heel and the forepart of the foot resist force to produce motion between the talus and the calcaneus. This condition is due to synovial reaction in the subtalar joint. It can be caused by strain. It frequently is the primary sign of rheumatoid arthritis. It can be due to a synostosis between the talus and calcaneus or between the calcaneus and the navicular bone (Fig. 82). These synostoses are apparent in special x-ray studies as a bar of bone or incomplete exostoses. Support of the foot with synostosis by means of arch supports, though occasionally of temporary aid, is not effective, and surgical stabilization of the talar-navicular and talar-calcaneal joints is necessary.

A painful foot occurs between the years of two and four. Indications

are the frequency with which the child removes his shoes and holds the arch of his foot. A limp will develop. Pressure in the arch of the foot, particularly over the navicular, gives discomfort. X-ray studies will show a narrow, dense navicular bone, which occasionally appears fragmented (Fig. 83, A). This is an *osteochondritis*, also known as Köhler's

Fig. 82. The synostosis between the talus and calcaneus is apparent in the oblique view.

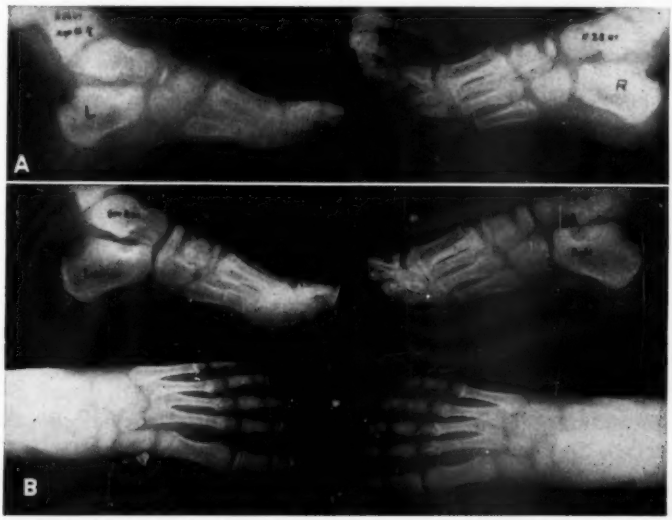

Fig. 83. *A*, The left foot shows the small, dense navicular (Köhler's disease) in a 4½-year-old boy. *B*, The same patient 20 months later. The navicular has healed and resembles that on the normal right side.

Fig. 84. Osteochondritis of the second metatarsal head of the left foot.

disease. The condition runs a self-limited course. It is not necessary to place the child in a cast. It is necessary to give the child a sturdy shoe with a longitudinal arch pad. He must also be cautioned never to put his foot to the floor without his shoe, even to go from bed to the bath. Regeneration of the navicular bone requires one to two years. The results are always good if the condition is seen early and the precautions conscientiously carried out (Fig. 83, *B*).

Freiberg's disease is an osteochondritis of the head of the second metatarsal bone. This appears from 10 to 12 years of age. The child complains of pain in the arch of the foot. By examination there is swelling about the second metatarsal head, and tenderness. X-ray pictures will show an enlarged head, a flat head, and at times a bony sequestrum within the joint (Fig. 84). Early, before sequestration, the use of an arch pad and low-heeled shoes are sufficient. Later the sequestrated fragment must be removed. In adult life removal of the head of the metatarsal is often necessary.

LOWER LEG

Deformities of the tibia vary from complete absence to apparent deformity from position of the foot or knee. With *congenital absence of the tibia,* the foot is displaced medially, the leg is shortened, and the distal end of the fibula is prominent at the ankle (Fig. 85). Preservation of the lower leg is necessary only if there is a proximal end of the tibia which may develop a useful knee joint. In bilateral absence of the

entire tibia, a child will walk on the ends of his femurs, dragging the useless appendages representing the lower legs and feet. A bilateral knee joint disarticulation is advisable in such cases. The child is then started with laced boots which are gradually elevated over the succeeding months until a height is obtained that will permit the use of prostheses with knee joints.

Tibial valgus or tibial varus may be observed in one leg of an infant or small child. An eversion or inversion of the foot is associated. An x-ray study will reveal an area of sclerosis where the angulation of the

Fig. 85. Bilateral absence of tibia with absence of toes and with "claw hands."

tibia occurs (Fig. 86, A). These children frequently have association areas of skin pigmentation or nodules as seen in congenital neurofibromatosis. The extremities are protected at once by means of a plaster of paris cast from foot to groin. Slight pressure is made to correct the foot position as the cast is applied. The cast is reapplied to continue correction at weekly and later monthly intervals. When growth permits, a brace is used on the leg to maintain correction and protect the tibia from fracture. A fracture or a corrective surgical osteotomy will result in a pseudoarthrosis that may resist all attempts at repair. The protective brace and a night splint are continued until the area of sclerosis has been replaced by a normal medullary canal and cortex (Fig. 86, *B*). The tibia will continue to be shorter than the one on the normal side.

Tibial varus (bowleg) is the most frequently observed position of the tibia under one year. This, as a rule, is self-correcting. A bowleg appearance persists if a club foot has not been completely corrected

Fig. 86. *A*, Tibial valgus in an infant. *B*, Same child after 4 years of plaster of paris casts and bracing.

or if an adducted foot is not corrected. The tibia in these cases may also appear rotated inward between the knee and ankle (internal torsion). It is therefore necessary, before making a diagnosis of tibial varus or tibial torsion, that any deformity of the foot or knee be considered and corrected first.

Rickets frequently results in bowing of the tibiae. This is recognized by a bilateral involvement and the other skeletal stigmata of rickets. The deformity in younger children is self-correcting if vitamin D intake is increased and activity permitted out of doors in the sunshine. Long leg braces are used if the bowing is 30 degrees or more. Rarely is it necessary or advisable to correct the bowleg deformity after the rickets has healed except in older children when growth is nearly completed and cannot compensate for the deformity.

Blount called attention to another cause of tibial varus which results in a premature arrest of growth on the medial side of the proximal epiphysis. This is unilateral. It is preceded by a beaklike projection of the metaphysis (not unlike that seen in rickets), followed by an area of rarefaction just beneath the epiphysis within the "beak" (Fig. 87). As growth continues uninterruptedly on the lateral side, the bowing of the leg increases. Upon early recognition, a brace is utilized to control the varus deformity of the knee. Later, as the condition becomes advanced, the growth of the lateral side of the epiphysis is stopped. An opening wedge osteotomy supported by a bone graft permits some

restoration of the plateau of the medial tuberosity of the tibia. Epiphysial arrest of the opposite tibia may also be necessary to prevent further inequality of leg length from growth.

Tibial torsion may result from rickets and from paralysis that produces an imbalance of leg musculature. There appears to be internal torsion of the tibia associated with club feet or adducted feet. As in tibial varus, its occurrence, while apparent, corrects with correction of the deformity of the foot. Rarely is surgical correction of tibial torsion necessary except in rachitic deformity or paralytic deformity. Then a rotation osteotomy of the tibia is done to correct the alignment of the foot with the knee. Toeing-in is frequently due to internal rotation of the legs at the hips.

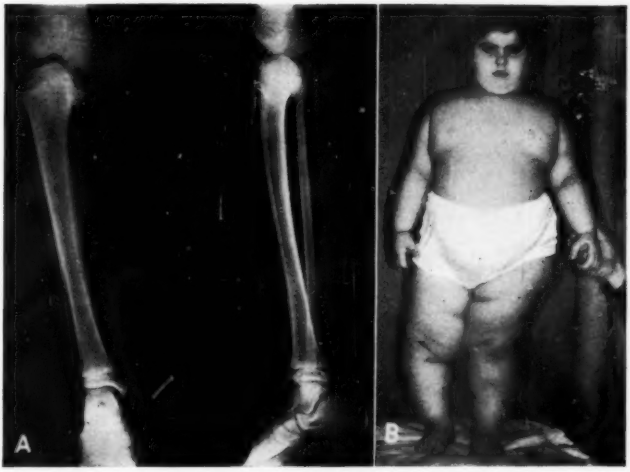

Fig. 87. A, Note beaking of tibia and arrest of proximal epiphysis on medial side. *B*, Note bowleg effect in child (obesity not contributory to condition).

KNEE

Deformity of the knee in infants usually accompanies other joint deformities. In *amyoplasia congenita,* or arthrogryposis, the knees are held stiffly in some flexion (Fig. 88). Associated deformities are club feet, dislocated hips, flexed wrists, extended elbows and restricted shoulder motion. Open inspection and biopsy of joints have shown no pathologic condition within the joints. The ligaments about the joints are somewhat thickened. The tendons are contracted, and the muscles are atrophic with replacement of fibers with fat and fibrosis. We are at a loss to explain the etiology. The outcome of these cases is most disheartening. The deformities are resistant to treatment. Although

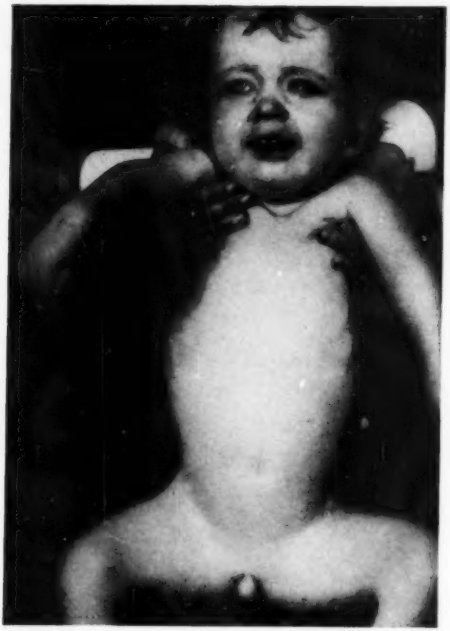

Fig. 88. Arthrogryposis. To be noted are the flexed wrists, stiff elbows and shoulders, dislocated hip and flexed knees. This child also had the associated club feet.

little improvement can be obtained by passive stretching, early splinting, or immobilization of joints to approximate a functional position, these measures in early childhood may facilitate the hoped-for result following operation as skeletal development matures.

Congenital dislocation of the knee is frequently bilateral (Fig. 89). The lower leg bends forward at the knee, and the knee cannot be flexed past 180 degrees. The lower leg can be lifted on the extended thigh for a distance of 45 to 60 degrees. The patellae, if palpable, are displaced to the lateral side. The tibiae are forward in relation to the end of the femur. The elbows of these children generally cannot be fully extended, since the heads of the radii are dislocated. Treatment of an infant with dislocated knees should be started at once to ensure normal development of the knee joint. Most cases respond readily to gradually increasing flexion of the knees, by repeated changes of cast at weekly intervals. Children who reach weight-bearing age without correction of the deformity stand with a noticeable back knee and are unable to flex their knees, so that their legs stick out in front or behind when sitting or standing (Fig. 90). Operation is necessary to reposition the tibia in relation to the femur. These patients never attain complete

Fig. 89. Infant with a dislocated knee characteristically holds leg over abdomen. The knee has no flexion.

Fig. 90. A 3-year-old girl with unreduced congenital dislocations of knees.

flexion of the knee, and generally have weakness of the quadriceps femoris muscle which necessitates a subsequent transplant of the tensor fasciae femoris and sartorius muscles to augment the power of the quadriceps femoris.

Dislocated patellae are generally bilateral. They are not recognized until the child is four or more years of age (Fig. 91, A). They are inherited. The family usually makes the diagnosis, since the mother or

father or some other member experienced the same condition. Otherwise they speak of the child falling and the knee going out sideways. When there is a unilateral dislocation of the patella, it is more apt to correct as the child attains maturity, unless it is due to a decided knock-knee (genu valgum), in which case bracing and development of the quadriceps femoris muscle may fail and operation is necessary to correct the deformity. A method of surgical correction of the dislocated patella depends upon transference of the attachment of the patellar tendon to a more medial position on the tibia and attachment of the semitendinosus tendon to the medial border of the patella to function as a check rein (Fig. 91, *B*).

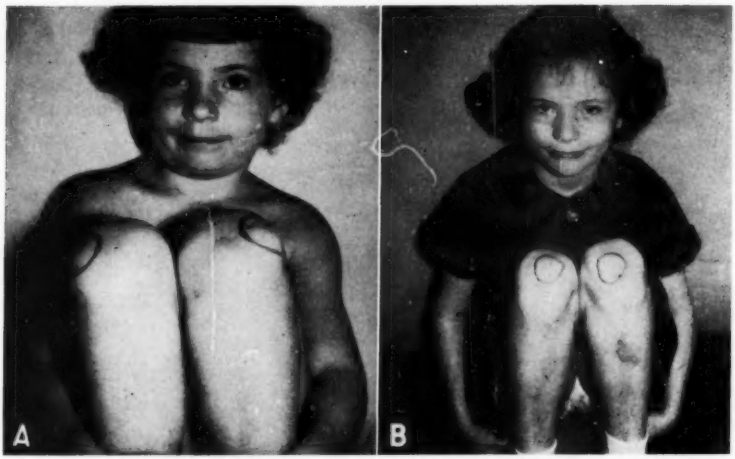

Fig. 91. *A*, Dislocated patellae. *B*, Several months after operation.

Baker's cyst of the knee is a ganglion or outpouching of the capsule in the region of the internal hamstrings on the back of the knee (Fig. 92). This pouching usually gives no difficulty in small children, and it tends to disappear. When it has persisted beyond the age of four and is associated with discomfort, it can be surgically removed.

Occasionally a cyst is observed in the region of the menisci. This cyst may be associated with either meniscus. Its size determines its removal, and the amount of meniscal involvement determines removal of the meniscus.

Osteochondritis is not uncommon about the knee joint. The most common site for involvement is the tibial tubercle. This occurs in children around 12 years of age. It is known as Osgood-Schlatter disease. Shortly before the tibial tubercle ossifies, it may be traumatized. It

may form spotty ossification and become extremely tender. If no treatment is instituted, a prominent tibial tubercle will result or ossified epiphysial fragments become scattered in and behind the patellar tendon. Children with Osgood-Schlatter disease complain of pain when kneeling and particularly in falls during athletic activities. They can be treated by means of knee pads to prevent trauma. Some response has been obtained with local infiltration of the area with hydrocortone. Temporary rest by means of a plaster cylinder for a period of six weeks gives improvement. In recurrent cases or in older children who are

Fig. 92. Cyst of knee commonly found along medial hamstring tendons.

strenuously athletic, surgically drilling multiple, tiny drill holes through the tibial tubercle into the underlying metaphysis is followed by prompt healing of the condition.

Avulsion of the tibial tubercle from a strong pull exerted by the patellar tendon may occur at 14 or 15 years. It is necessary to fix the tibial tubercle with a metallic screw or wire suture.

Next in order of involvement is the medial condyle of the femur. This again becomes apparent by about 12 years of age. The child complains of pain with activity, has some swelling of the knee, and develops slight limitation of motion. X-ray study to show the notch between the condyles of the femur will reveal an area of rarefaction

about a peripheral area of irregular calcification on the lateral side of the medial condyle. Protected weight bearing and rest from strenuous activities for a period up to four years generally result in healing of this condition. Occasionally in latent cases there is a sequestration of the area of osteochondritis. This forms a loose body (*joint mouse*) which becomes locked within the joint and must be removed.

By about 14 years of age one or both knees may become painful and develop periodic effusion, particularly after running, playing tennis or climbing stairs, and there is audible grating beneath the patellae. Rest alleviates symptoms, but they promptly recur as activity is increased. X-ray studies of the patella will reveal a central crater on the posterior surface, surrounded by a sclerotic margin. The treatment of this condition is by limitation of activities and restriction of athletics. Occasionally there is sequestration of a button of cartilage-covered, calcified material from the defect in the patella. This must be removed from the joint.

FEMUR

Fracture of the femur in infancy follows a difficult delivery. It is recognized as the leg lies in external rotation. The child does not move the extremity. The thigh appears thicker and shorter than that of the opposite leg. If the leg is lifted, the infant will cry, and there is bizarre motion due to the "jointed" thigh. No attempt should be made to reduce a fracture of the femur in a young child. It is important to stress that this leg be immobilized in neutral position; i.e., place the foot, knee and hip in the same position as in the opposite leg and apply a hip spica (Fig. 93). After six weeks of age a double spica is used. This spica can be cut out sufficiently to permit a folded diaper to

Fig. 93. Birth fracture of the femur in an infant is held in plaster spica with hip and knee in flexion. Cast may be supported by a pillow. When infant is prone, a pillow is used under chest.

be placed over the perineum. The head of the crib is elevated so that there will not be soiling by urine or feces beneath the cast. Fractures of the femur in infants should not be treated by means of Bryant's traction. Bryant's traction is a method of treating fractures of the femur in small children by suspending the leg from an overhead bar or pulleys on the crib. In infants the circulation can be impaired by fully extending the knee, since it places a stretch on the popliteal artery.

Fig. 94. Child with a congenital short left femur.

Avulsion of the distal femoral epiphysis may occur during delivery. This will produce swelling of the knee. Its recognition may be difficult even by x-ray examination. Reduction of this fracture is not necessary or advisable. Immobilization as shown in Figure 93 results in remodeling by the periosteum of the proximal fragment of the femur and no residual deformity.

Congenital short femur also gives a strange appearance to the leg (Fig. 94). The leg is short, and the thigh is thicker than the opposite one and is considerably shortened. It has a resemblance to a leg of a lamb. The motions of the hip are restricted, particularly in abduction, internal rotation, and extension. X-ray study will show an apparent

absence of the upper end of the femur with a telescoped normal distal two thirds and knee joint. The absence is due to lack of ossification of the upper end of the femur rather than complete loss of substance. The actual gross appearance of the proximal femur is that of an irregular cartilaginous mass bowing anterolaterally. The head of the femur therefore is in varus position. Ossification does not take place until seven or eight years of age, and the deformity is permanent. Early management is by a nonweight-bearing, caliper brace, later osteotomies to correct the femoral deformity, epiphysial arrest on the good leg, and finally an elevated shoe to balance the remaining leg inequality.

HIP

Dislocation of the hip in an infant is recognized by the lack of abduction of the hip (Fig. 95). This is generally noticed when diapers are

Fig. 95. Demonstrating a characteristic sign of dislocation of the hip.

changed. The extra wrinkle in the thigh sometimes is of significance. The crepitus in the hip with motions in flexion-extension or rotation is a determining sign. Double dislocations of the hip are somewhat more difficult, but again should be suspected because of lack of abduction, the crepitus on motion, the breadth of the hips, and a spreading in the perineal region rather than a tight crease. As the child becomes older, additional signs become more apparent such as the lack of internal rotation of the thigh, the higher position of the greater trochanter, the piston mobility of the hip. (If the examining hand is placed on the trochanter and the foot is pulled and pushed, the trochanter will glide under the examining hand.) In children of weight-bearing age the positive Trendelenburg sign and the waddling gait are indicative signs.

Another cause of waddling gait in a child is the *coxa vara* deformity

of the hip (Fig. 96). This means that there is a decrease in the angulation of the neck of the femur so that the abductor muscles of the hip span a shorter distance and lose their tensile strength. As the child steps upon the involved leg, there will be a drop of the pelvis on the opposite side, producing a waddling gait. The coxa vara deformity is congenital. It is corrected only by surgery, which produces a valgus position of the neck in relation to the femoral shaft.

Between four and eight years of age, with upper limits of 10, children frequently will develop a limp, sometimes night cries, and some limitation of hip motion on examination. The motions in extension and

Fig. 96. The x-ray film of a 7-year-old boy with bilateral coxa vara.

abduction are particularly limited. Early x-ray examination may reveal nothing more than a slight increase in density of the femoral head. Later examinations will give an appearance of fragmentation of the femoral head or flattening or absorption of the epiphysis (Fig. 97). This condition is commonly known as *Legg-Calvé-Perthes disease*, or osteochondritis of the capital epiphysis of the femur. Early recognition followed at once by protection from weight bearing on the femoral head (capital epiphysis) will result in restitution of the femoral head without deformity and without any loss of motion or fear of degenerative changes in the hip in later years. This restitution of the femoral head takes from one to four years. It is not unusual for the opposite hip to become involved while the other hip is being treated. If the x-ray films show deformity of the capital epiphysis on the first examination prior to restricting the child's weight bearing, little hope can be main-

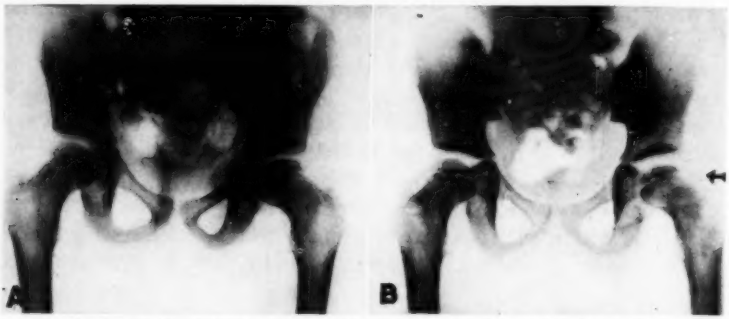

Fig. 97. Early diagnosis of Legg-Calvé-Perthes disease (*A*, right hip) is frequently difficult, but should be suspected if there is a variation in bone density or diminution of size of the epiphysis of the head of the femur (*B*, right hip taken 6 months previously).

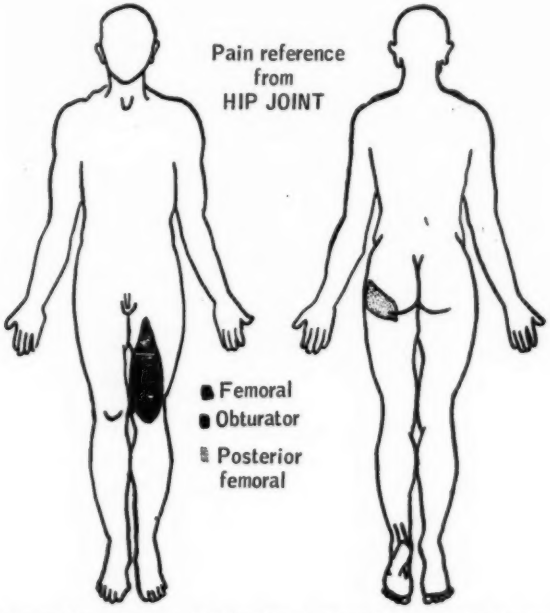

Fig. 98. Diagram showing nerve distribution of referred hip pain in slipping of capital epiphysis of femur.

tained for complete restitution of the height of the femoral head. The result, therefore, will be impaired unless treatment is started early.

Slipped capital femoral epiphysis occurs in girls from 11 to 13 years of age, but more predominantly in boys from 12 to 14 years of age. This condition may have a sudden onset, but more generally, in obese

adolescents, it is associated with pain provoked by activity over several weeks or months. The difference in leg length is slight. The lack of hip motion can be determined only by comparing the motion of the opposite hip. The pain reference is usually to the knee (Fig. 98). X-ray film of the knee may show at this age fragmentation of the tibial epiphysis, so that not infrequently the patient is treated for Osgood-Schlatter disease while the deformity of the hip increases. In those

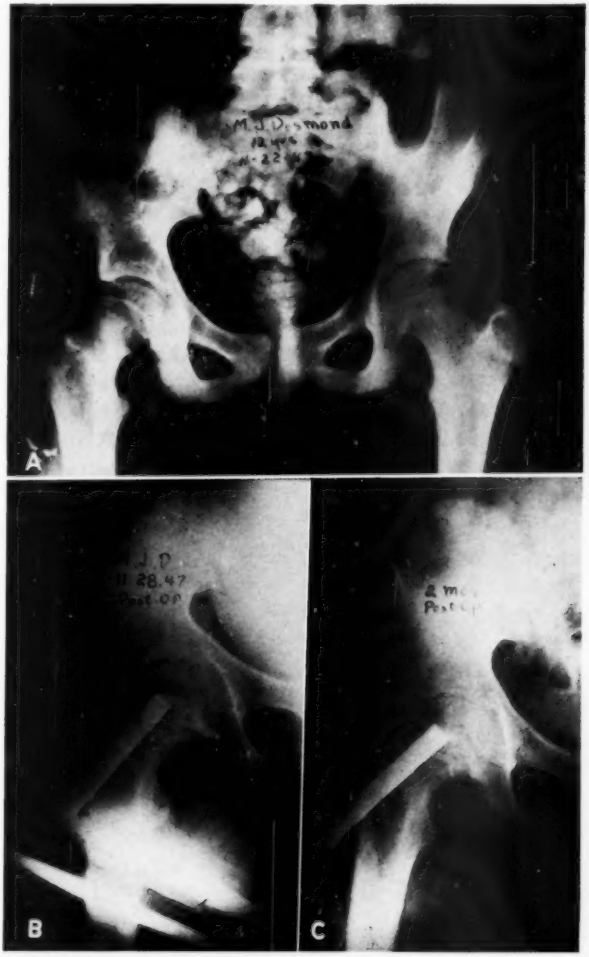

Fig. 99. A, Slipped capital femoral epiphysis on the left. B, Restoration of position of head after osteotomy. C, Two months after operation the head shows increased density indicative of circulatory embarrassment.

that have a sudden onset in an athletic endeavor, the deformity is greater, the pain is acute, and weight bearing is impossible, since the epiphysis is separated from the femoral neck. Recovery from a slipped capital epiphysis is entirely dependent upon early recognition. The lateral x-ray view of the hip facilitates the diagnosis, since the deformity occurs as the neck of the femur slides upward anteriorly, displacing the femoral head backward. If the condition is recognized and cared for before the slip is more than one third of the distance from its normal position, the result will be good. In those with a complete slip or in whom reconstruction must be undertaken to correct the deformity, a disturbance of the circulation of the head may result in avascular necrosis and permanent deformity of the hip joint (Fig. 99).

Arthrokatadysis (Otto's pelvis) is recognized at adolescence. The hip motion becomes limited, particularly in abduction and in rotation. The child develops a limp. There may be some pain in the groin or referred to the inner side of the knee. X-ray studies confirm the diagnosis by showing an abnormally deep acetabulum with inclusion of the head of the femur in the deepened hip socket. Although this condition cannot be cured, rest with traction on legs and no weight bearing or protected weight bearing if unilateral, until the tripartite epiphysis of the acetabulum has united, must be observed. When the floor of the acetabulum has become completely ossified, there will be no further increase in the deformity until disability or age results in osteoporosis or degenerative arthrosis. Complete motion of the hip will not be re-

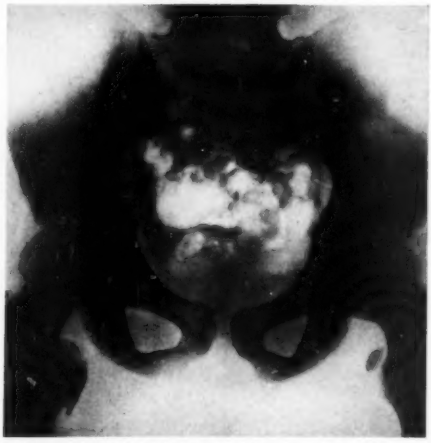

Fig. 100. An avulsion of the right lesser trochanter of the femur from iliopsoas pull. The identical injury occurred on the left side one year previously, and healed with bony proliferation, but with no impairment of function.

gained. Surgical procedures for correction of the deformity should be postponed until adult life.

Another cause for sudden, sharp pain about the hip region in adolescent children is *avulsion of an epiphysis* to which a tendon attaches (Fig. 100). This occurs during athletic competition, particularly games which require running and twisting activities of the body. The most common avulsions of tendon attachments are the inferior-anterior iliac spine from the rectus femoris, the lesser trochanter of the femur from the iliopsoas, and the ischial tuberosity from the biceps femoris. The epiphysial attachments of the rectus femoris, iliopsoas or biceps heal spontaneously with bed rest or a plaster of paris hip spica for six weeks. This results in some increase in size of the bony attachment, owing to proliferation of new bone. A differentiation of the x-ray appearance from a bone tumor is frequently based upon an accurate history.

SPINE

Spinal curvature, or *scoliosis*, rarely is apparent in an infant without an anatomic variation such as a hemivertebra, spina bifida occulta, fused vertebrae, and rib deformities. X-ray study determines the nature of the deformity. Infants with a structural or an idiopathic curvature are placed in a plaster bed which holds their spine bowed to the opposite direction from which their curvature is directed. Through growth, frequently it is necessary to maintain the overcorrected position for the scoliosis by means of corsets, braces, celluloid jackets, special beds, until the child has approached adolescence. At this time it is possible to perform surgical fixation of the spine with minimum possibility of the stress of future growth producing a fracture, a pseudoarthrosis, and recurrence of the deformity. Although attempts were made in the past to remove hemivertebrae in order to diminish the deformity, these resulted in increased deformity during growth as well as a kyphos deformity at the area from which the hemivertebra was removed. As all cases of hemivertebra have a tendency to kyphos deformity in addition to scoliosis, a not infrequent complication is paralysis. This is due to the bowing of the spinal cord over the deformed kyphos area. A laminectomy is of no avail in relieving the paralysis. It is necessary in these cases to decrease the deformity with extension of the spine and/or relocate the cord outside of the spinal canal on the concave side of the kyphoscoliosis. This is followed by surgical stabilization of the spine at the site of deformity.

Juvenile kyphosis, or preadolescent kyphosis, occurs at the age of eight or nine years. The thoracic spine becomes rounded, and the

child develops "round shoulders." These children are apt to be rapidly growing, more or less atonic in musculature, and less athletic than others. At this age it may be called the "television spine," since many young children spend several hours a day crouched forward in a chair with their elbows on their knees, a finger in the mouth, or their chin in their hands, biting their nails while watching the screen before them. Occasionally collapse of a vertebra in the thoracic area is noted. This was called in the past *Calvé's disease*. This vertebral collapse was believed to be due to an osteochondritis. In recent years it was found that *eosinophilic granuloma* will cause such collapse of vertebrae. Of course it is well known that *tuberculosis* will result in such a vertebral deformity. A differential diagnosis therefore must be made in these cases. Early management with rest and moderate extension is the same in all three conditions until the etiology is determined. In the juvenile kyphosis, Calvé's disease or the eosinophilic granuloma the treatment is relatively the same, with a hard, flat bed. Television hours are spent lying prone on the floor, propped on elbows. The same position is utilized for reading, writing, study. Exercises to develop the upper back muscles, such as head- and arm-raise from the prone position, use of chin bar, and push-ups are recommended. Supportive braces are used as reminders, but actually give little support to the upper spine unless the head is included in the brace. The eosinophilic granuloma, in addition, receives x-ray therapy. In tuberculosis of the vertebrae, of course, the focus of infection must be identified and treated. If an abscess has formed at the spine lesion, it is drained. The child is treated with streptomycin, para-aminosalicylic acid and isoniazid for 6 to 12 months. The child should be observed carefully for signs of developing paraplegia, and, when the acute situation is controlled, surgical fixation of the vertebra to its neighboring vertebrae should be undertaken.

Before a diagnosis of *idiopathic scoliosis* can be made, other causes of scoliosis should be determined. It is necessary that a complete neurologic examination, a muscle test to determine muscle imbalance of the trunk or thigh muscles, measurement for leg inequality, and x-ray examination of the back for vertebral or rib deformity be completed. Although idiopathic scoliosis may develop as early as 6 years, it generally is not recognized until 11 or 12 years, when spinal growth becomes more rapid. Approximately 80 per cent of these cases are in girls. There appears to be a familial tendency. Identical twins, more than one child in a family, and not infrequently a parent and a child will have scoliosis (Fig. 101). Posture plays a roll in the development of scoliosis. Every endeavor to maintain as erect a spine as possible during the childhood and rapid growth years of preadolescence must be made. Exercises to develop the spine, abdominal and thigh musculature should be carried out routinely. The habits of the patient must be

Fig. 101. Idiopathic scoliosis in identical twins. The girl on the left had, at a later date, correction and surgical stabilization of the deforming thoracic curve. The girl on the right will not have operation, since the lower thoracic and lumbar curves give little disability or deformity.

policed rather closely. Study periods at home should be spent lying prone on the floor propped on elbows. The desk chair must be straight, with a right-angled back and seat; it must fit beneath the desk so that the subject's back is flat against the back of the chair and the height of the desk comes to the level of the elbows when the arms are flexed across the chest. The bed must have a thin mattress, 2 or 3 inches thick, without inner springs. Beneath the mattress must be placed a board 5/8 inch in thickness. There should be no pillow. Any tendency to relax posture habits or show increase in deformity of the spine must be met with support such as corset, brace, celluloid jacket, or recumbency. Early operation has been disappointing because of the tendency for the deformity to return as activity and the force of gravity were permitted to lay stress upon the spine. The ideal time for operation is when but two years of spine growth remain. A rough estimate of this can be made from x-ray studies which show beginning ossification of the rim of the pelvis. Important after surgical fixation is a prolonged period of immobilization in a cast from occiput to pelvis for a minimum of one year (Fig. 102).

Spondylolisthesis occurs in young children. It is a congenital failure of ossification of the posterior processes of the fifth and less often the fourth lumbar vertebrae. As a result of this failure of ossification, there is an insecure fixation to the subjacent vertebral processes. This results

in an anterior displacement of the vertebrae involved. Of course the superjacent vertebrae follow the course of the involved vertebra. The displacement is frequently progressive. These children seldom complain of backache, but are particularly prone to tire on standing. They characteristically sit when they stop walking. On examination their buttocks are prominent, and there is shortening of the trunk between the ribs and the iliac crests. In the more obese there is a wrinkle across the back above the iliac crest. Upon palpation the sacral spinous processes are prominent, and, as the examining finger is moved upward on the back, there will appear to be a depression of the lumbar spinous

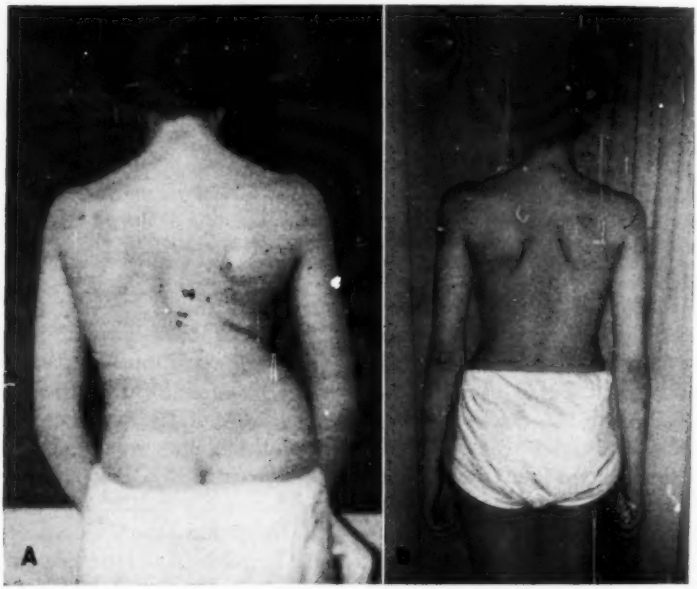

Fig. 102. Shows the cosmetic result that can be obtained in selected cases. *A*, Before operation. *B*, Eighteen months after operation.

process above the sacrum. The erector spinae muscles will be prominent on either side. There will be a hollow between these muscle masses above the sacrum. Occasionally there will be neurologic signs such as ankle clonus, some weakness of the dorsiflexors, and diminished sensation in the perineal area. Younger patients without neurologic findings who by x-ray examination show less than 25 per cent forward displacement of the vertebra on the subjacent vertebral body frequently do well with bracing, restriction of activities such as body contact sports, heavy lifting, the use of straight-back chairs and a hard, flat bed. In older children who are athletically inclined and have more than one

third forward displacement of the vertebra, surgical fixation of the spine should be done.

CHEST

The most common deformity of the thorax is that due to *rotation of the ribs* from a thoracic spinal curvature. Management is by correction of the curvature. The correction of a lateral deviation of the thoracic spine does not result in an appreciable correction of the rib rotation. To date there has been no satisfactory surgical procedure devised which will result in any great improvement of the rotation deformity of the thoracic cage. Deformity is also due to congenital absence of ribs or congenital fusion of ribs. The sternal deformities consist of the *pigeon breast*, which is caused by rickets. This may be prominent in younger years. As adolescence is achieved, the deformity is less conspicuous, owing to muscle or breast development. Since there is no embarrassment of respiration or cardiac function with such deformity, surgical procedures should be deferred, for they are seldom necessary from a cosmetic standpoint.

Pectus excavatum is apparent in young children. There is a depression of the mid-portion of the sternum (gladiolus) (Fig. 103). This depression is influenced by a band attaching from the diaphragm. The tip of the sternum (xiphoid) projects anteriorly and is prominent. Laterally the costochondral junctions are angulated sharply, producing a margin to the bowl-like deformity of the sternum. This deformity can severely crowd the heart, and in adult life or in athletic persons can be

Fig. 103. Pectus excavatum in a 12-year-old boy of small stature and a spinal curvature.

responsible for cardiac embarrassment. Although some success in infancy has been accredited to division of the sternal attachment of the diaphragm, the operation in older children necessitates elevation of the sternum by corrective osteotomy at its junction with the manubrium and the xiphoid and of the ribs lateral to and at the costochondral junctions on the sides. Postoperative treatment is somewhat prolonged, since it is necessary to suspend the sternum from an out rigger in order to prevent its collapse into a position approximating that before operation. The result to be obtained from operation for pectus excavatum requires considerable patience as well as technical skill on the part of the surgeon and cooperation on the part of the child and his parents.

UPPER ARM

As the newborn infant is examined about the shoulders, a *fracture of the clavicle* may be determined. As a rule this is not a serious complication. The clavicle will heal rapidly. There will be a mass of callus within a few days. Even with considerable overlap of the fracture fragments, there will be no shortening of the clavicle after six months of growth. For the child's comfort during the first few days, a binder is placed to hold the humerus and the arm against the chest. It is unnecessary and inadvisable to use a figure-of-eight bandage on an infant with a fractured clavicle.

A *brachial palsy* frequently accompanies a fractured clavicle. This will be recognized, since the child will not abduct its arm, but will hold the arm in internal rotation with the forearm in pronation so that the back of the hand is against the thigh. The brachial palsy can be readily missed in the presence of a fractured clavicle, since the finding of the fracture may deter the examiner from looking further. A brachial palsy, so called, is due to a stretch of the brachial plexus of nerves, but may be due to an actual tear of the nerve roots in the cervical region. If the latter has occurred, there is little chance of recovery. At the time of early examination it is not possible to determine in infants the level or the extent of the injury. If, however, the lower arm appears to be involved and there is little response to pin prick in the fingers or about the hand, the chances of more severe injury should be suspected, particularly if the child shows a Horner's syndrome. The deforming feature in a brachial palsy is the contracture of the uninvolved muscles (Fig. 104). Within a few days, therefore, after birth a plaster bed is made for the infant which will permit the shoulder to be held in abduction, external rotation, and the forearm to be held in supination, with the hand and fingers extended. The fingers are never to be hyperextended. If there is an accompanying

Fig. 104. A brachial palsy of the upper arm type in a 3-year-old boy who has had no treatment.

fracture of the clavicle or of the humerus, this position of the arm in the plaster bed will not influence in an adverse way the healing of either fracture. The plaster bed should be utilized at all times, even when feeding the infant. After the first week the infant may be removed from the bed for bathing purposes. It is generally necessary for the plaster bed to be continued for three to four months. At that time it is possible to fit the child with a brace. If the brace is to be continued for more than six months, it is necessary that the child have periods of freedom from the brace or contractures will develop which maintain the arm in abduction and external rotation position. It is therefore advisable that the brace be removed and utilized only for hours of sleep, during the nap time and night time. Surgical procedures are seldom indicated before two years of age, at which time a maximum return of motor and sensory nerves can be expected. In the years before growth of the humerus is completed, only release of contracted tendons should be considered, and transplant of the tendons to increase the power of external rotation and abduction of the shoulder.

Cleidocranial dysostosis may be determined in infancy by the narrowed appearance of the chest. The shoulders can be brought forward as the child is lifted by the arms so that the deltoid areas of the oppo-

site shoulder are brought into approximation. The head frequently has a flattened appearance in the anteroposterior direction (Fig. 105). The cranial bones, particularly the frontal, parietal and occipital, are small, with wide sutures which have delayed union. There is also delayed ossification of the symphysis pubis, delayed growth of the long bones, and

Fig. 105. Cleidocranial dysostosis. (Courtesy of Dr. Robert D. Heath.)

usually scoliosis. There is also delay in loss of deciduous teeth and eruption of permanent teeth.

Sprengel's deformity (congenital elevation of the scapula) is frequently accompanied by changes in the cervical spine such as hemivertebra or reduction in the number of vertebrae (Klippel-Feil syndrome) (Fig. 106). The deformity may involve one scapula or both scapulae. The scapula is small. It is pulled toward the midline and is prominent above the base of the neck. The elevation of the arm on the involved side is considerably limited by the lack of abduction of

the shoulder. By x-ray film an omocervical bone may be apparent in some cases. This is a bone which has a fibrous union with the base of the spinous process of the scapula and to either a spinous process or transverse processes of the cervical spine. There is no early treatment of the deformity. Operation, however, can be undertaken at six months of age. It consists in freeing the muscles that elevate the scapula, removing the omohyoid bone, and resection of the prominent supraspinatus portion of the scapula. Traction by means of a wire through

Fig. 106. This 2-year-old child has a Klippel-Feil syndrome, reduction and fusion of cervical vertebrae, and a Sprengel's deformity, congenital elevation of the left shoulder.

the lower scapula attached to its spinous process is maintained through a turn-buckle or elastic attached to a plaster of paris hip spica. Although a normal shoulder and normal-appearing scapula are not obtained, the function of the shoulder and the cosmetic effect are considerably improved.

ELBOW

The elbow joint may be fixed in extension in arthrogryposis. The most common lesion of the elbow is probably *dislocation of the head of the radius.* This is anterior. It may limit complete extension of the elbow joint. It may accompany dislocated knees. It is seldom recognized in

infancy. Improvement in function is not obtained by surgical reduction. In older children the dislocation gives relatively little disturbance; therefore correction has not been undertaken except in acute dislocations.

Traumatic subluxation of the radial head occurs in small children as a result of their being lifted or supported by one arm. It has been termed "curb-stone elbow" because of its frequent occurrence while helping a child over a curb. Characteristically the child screams, lets his arm dangle, refuses to permit anyone to touch the arm. The forearm is usually fixed in pronation. As the child is calmed, the physician will slowly and gently flex the arm by grasping the wrist with one hand and the elbow with the other so that his thumb will be placed anterolateral to the head of the radius. With mild pressure of the thumb on the radial head, while the forearm is slowly supinated and pronated, a click is heard or felt and the child is restored. Anesthesia is seldom indicated. A sling may be used until the child discards it.

FOREARM

Congenital absence of the radius is accompanied with a club effect of the hand in which the hand is turned laterally at a right angle and displaced toward the elbow. The end of the ulna is prominent beyond the wrist (Fig. 107). Early management is best accomplished by means of a plaster cast in order to hold the hand in a nearly normal position in relation to the end of the ulna. Later, molded plastic cuffs are utilized to maintain the position of the hand, and at 18 months or 2 years

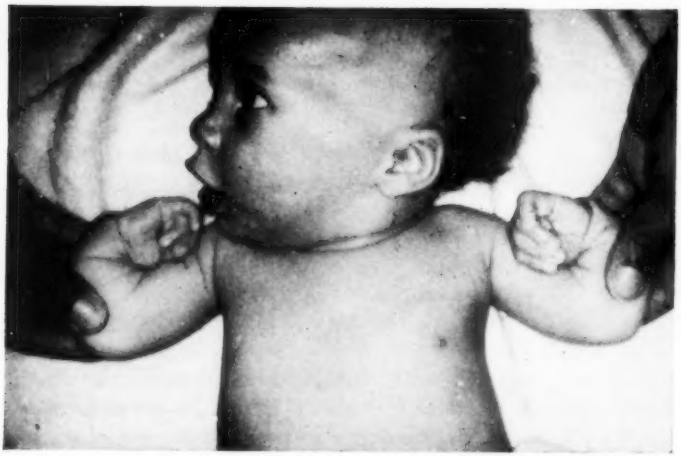

Fig. 107. Bilateral congenital absence of the radius in an infant.

of age the capsular structures are released about the carpal bones and the hand is approximated over the end of the ulna. It is maintained in this position by means of small pull-out Kirschner wires. At a subsequent date a bone block is substituted for the absent radius. The best method is that of Roidon, utilizing the epiphysis of the proximal end of the fibula and grafting it upon the ulna to act as a block to prevent radial deviation of the hand. Although the fibular epiphysis does not continue to grow in this position, the block is effective and can be moved distally at a later date as radial deviation tends to recur.

Fig. 108. Congenital absence of the ulna (right in photograph) results in a useless forearm.

Congenital absence of the ulna is more deforming (Fig. 108). It is not uncommon for the thumb alone to articulate through its first metacarpal, greater multangulum and navicular to the radius. The radius is always dislocated at the elbow and is held in flexion so that the forearm is of little use. These cases are benefited by early amputation through the elbow and application of a prosthetic device.

Fixed *flexion of the thumb* is frequently overlooked in examination of the infant (Fig. 109). This is not unlike a trigger finger in an adult. At times it can be pulled free. Most generally the "pulley" is so thickened and there is such a large nodule in the long flexor of the thumb that extension cannot be accomplished. The simple procedure of making an incision on the palmar side in the skin crease at the base of the

Fig. 109. Congenital fixed flexion of the right thumb in an infant.

thumb and splitting longitudinally the "pulley" over the flexor tendon results in complete cure of the condition.

Early evaluation of musculoskeletal lesions permits the pediatrician and the family to reach an understanding of a problem that can be best corrected primarily or secondarily, throughout growth or after growth is completed. Recognition of and surgical consultation for a deformity will result in better preparation of family and child for the timing of the surgical procedures as they are indicated.

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COMMON MALIGNANT TUMORS OF INFANCY AND CHILDHOOD

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Current statistics indicate that malignant disease in the United States is the second most common cause of death in childhood. It is exceeded only by accidents. Because present methods of treatment hold some promise for many of these children, it is important to be aware of the characteristics of the types of malignancies seen before puberty. A greater understanding of the natural history of these conditions will enable the physician to plan effective methods of management.

In general the group of tumors to be considered here is peculiar to infancy and childhood. On the other hand, it is important to recognize that most of the malignant diseases in adults are of epithelial cell origin (carcinoma). Carcinoma comprises 90 per cent of the malignancies following puberty, whereas, in a series of childhood malignancies recently studied in Seattle, carcinoma comprised only 4 per cent of the total.[1]

Some malignancies are present at birth or are discovered within a

From the Tumor Clinic of the Children's Orthopedic Hospital, Seattle, Washington.

few months of birth, indicating intrauterine development. Others, such as retinoblastoma, are genetically determined. Malignant mesenchymal tumors generally metastasize through the blood stream, but may also spread by way of the lymphatics.

To simplify this discussion, the childhood malignancies are classified according to the type of neoplastic tissue rather than by anatomic location. In our series of 408 cases the types of malignancies were found to occur in order of frequency shown in Table 3.

TABLE 3. *Incidence of Childhood Malignancies**

TYPE	CASES	PER CENT
Leukemia	124	30.5
Tumors of the central nervous system (including retinoblastomas)	80	19.5
Tumors derived from the adrenal medulla and sympathetic nervous system (neuroblastomas and allied tumors)	43	10.0
Lymphatic tumors (not including leukemia)	31	8.0
Tumors of the kidney (Wilms's tumors)	31	8.0
Bone tumors	26	6.0
Soft tissue sarcomas	25	6.0
Carcinomas	18	4.0
Teratomas	15	4.0
Miscellaneous tumors	15	4.0
Total	408	100.0

The same order of frequency has been found in similar series studied elsewhere.

* Children's Orthopedic Hospital and Tumor Institute of Swedish Hospital, Seattle, [1] 1949–1958.

It is important to recognize that certain types of malignancies will not be seen in infancy and childhood. For example, carcinoma of the breast before puberty has been reported in less than 10 cases. Carcinoma of the skin is so rare as to be a medical curiosity. To our knowledge carcinoma of the lung has not been reported. Carcinoma of the alimentary tract occurs rarely in the colon. Malignant melanoma is extremely unusual.

With this as a background we shall consider individually the malignant tumors usually encountered in infancy and childhood, with the exception of leukemia and central nervous system tumors.

NEUROBLASTOMA

Behavior and General Characteristics

Because of its frequency neuroblastoma holds an important place among malignant tumors in childhood. With modern methods of treatment the possibility of a cure has been reported as being approxi-

mately 25 per cent. This tumor is believed to hold a unique place among all human malignancies in one particular aspect: it will sometimes change from malignant to benign by maturation of the cells to a ganglioneuroma. At times the neuroblastoma seems to regress entirely.

Neuroblastomas arise from the adrenal gland medulla and the ganglia of the sympathetic nervous system. The greatest number originate from the adrenals, but these tumors have no endocrine function. Fifty-seven per cent appear in the abdomen.

Since the tumor may originate anywhere along the chain of sympathetic ganglia, it may appear in the chest in the paravertebral region, in the neck, and as low as the bifurcation of the aorta. Although the primary tumor may cause the first clinical signs or symptoms, a fairly large proportion of these neoplasms will be first noticed through metastases. The early signs of neuroblastoma may be from metastases to bone, to the skin or to lymph nodes. When there are metastases to bone, these may appear as a mass growing from some area such as the skull, or they may cause pain or disability, as in metastases to the femur or pelvis. Occasionally the first sign of the tumor will be a mass appearing in the back, which will have extended directly posteriorly from the original site in a sympathetic ganglion. The manifestations of neuroblastoma, therefore, are protean, and the possibility of the disease must be kept in mind in many conditions which appear in early life.

In some young infants with this disease the first sign will be from massive infiltration of the liver, without evidence of the primary origin. In significant numbers of these young patients the liver has regressed to normal size and the infants have survived to a normal life. This has occurred most frequently after x-ray therapy, but occasionally without it.

The prognosis is almost universally bad in the event of metastases to bone. The prognosis is better the younger the infant, and also when the lesion is found in pelvic or cervical areas. It is excellent when the lesion is localized and can be removed intact.

A patient with a tumor in the region of the kidneys should be suspected of having a neuroblastoma. When examined by intravenous pyelogram, a neuroblastoma will be seen to displace the kidney by extrinsic pressure, with the direction of displacement dependent on the location of the primary and the mass of local lymphatic spread. In order to demonstrate the fine speckled calcification which is almost pathognomonic of this tumor (Fig. 110), films must be obtained without any motion. Rapid exposures of 1/30 second can be made on infants with the standard Bucky set at 1/10 second. Urograms rarely show distortion of the calyces or pelvis. Extensive caseous metastases with characteristic mottling of the calvarium suggest this diagnosis; therefore skull films and general skeletal survey are important in differentiating Wilms's tumor, leukemia and reticulosis.

Fig. 110. Neuroblastoma. Large mass, left adrenal region demonstrating the almost pathognomonic calcification. Kidney displaced, but not distorted.

Treatment

The primary tenet of the physician treating a patient with neuroblastoma is never to give up trying! This is true in this disease as in no other malignancy, since it may reverse its course if its progress can be kept in check long enough.

Surgical excision of a neuroblastoma should be undertaken whenever possible. The ideal is total excision. When the tumor is located in the region of the adrenals, the surgical approach should be through the abdomen. When a neuroblastoma is not totally resectable, it is presently believed that as much as is consistent with the safety of the patient should be removed. Preoperative irradiation may be of assistance in such a partial resection when it is recognized before operation that total removal will be impossible. In such cases preoperative x-ray therapy will help by shrinking the tumor and by decreasing its vascularity.

Although this disease is frequently widespread when diagnosed, radi-

ation therapy is indicated. Reports in the literature describe apparent cures with doses as small as 400 roentgens delivered to the tumor. X-ray therapy should start the day of operation, the tumor dose to be carried to at least 1200 roentgens in air. Recent evidence shows that reduction in tumor size by operation makes its response to irradiation greater.

Wittenborg[12] states that the limiting factor in treatment lies in damage to the hematopoietic system and not to the epiphyses. Swenson[11] believes that the treatment of bone metastases by irradiation is not effective. Although this may be true from a curative standpoint, judiciously applied irradiation will give considerable symptomatic relief.

Of the chemotherapeutic agents, nitrogen mustard is most frequently used. As in other malignancies, it is an adjunct for palliation. Actinomycin-D has proved useful in potentiating the effects of irradiation. The use of large doses of vitamin B_{12} has been advocated by Bodian,[2, 3] who has reported encouraging results with the use of this drug. These results have not yet been duplicated in the United States. In advanced stages of the disease, particularly with hemorrhage, adrenal hormones can offer the patient a great increase in comfort.

LYMPHATIC TUMORS (LYMPHOMAS)

Behavior and General Characteristics

Any unusual lymph node enlargement in a child should be observed with suspicion. Lymphatic tumors, excluding the leukemias, are the fourth most frequently observed of the malignant growths in childhood. Of these, lymphosarcoma is the most common and yields the best prognosis. Hodgkin's disease and reticulum cell sarcomas are related lesions and are more malignant. The diagnosis of any one of this group lies in the study of an adequate biopsy. These tumors are found primarily in children over 3 years of age, with a peak incidence from 8 to 14 years.

In over 95 per cent of these patients lymph node enlargement is the presenting sign. Tonsillar and adenoidal overgrowth, pulmonary hilar enlargement, splenomegaly, bone pain, unexplained fever, or anemia, infiltrative skin lesions and, rarely, central nervous system symptoms may also be present. In all these children the diagnosis should be sought through lymph node biopsy or bone marrow examination. Approximately 20 per cent of children with lymphosarcoma suffer leukemia as a terminal stage.

Treatment

With the realization that many lymphomas are representative of general disease, the place of surgery is limited. There are, however, certain

cases in which the lesion is localized to the neck, axilla or gastrointestinal tract. In such instances complete surgical removal should be attempted and may be successful.

With few exceptions all the tumors included in this group are radiation-sensitive, but not curable. Some, occurring as localized lesions, may respond with occasional cure to irradiation as well as to surgery. When the disease has become disseminated, x-ray is used only for local palliation. It is interesting to note that in this disease response to nitrogen mustard parallels the response of the tumor to irradiation. If either modality is effective, they will be about equally effective, and when either becomes ineffective, both will be ineffective. When the disease is localized and treatment is given in an attempt to cure, the x-ray dose should be in the order of 2500 to 3000 roentgens to the tumor. When treatment is given for palliation, smaller doses may be sufficient.

In this group of tumors medical treatment combined with irradiation is useful for palliation. Rosenberg[10] recently reported the largest experience with treatment of lymphosarcomas, which includes local irradiation to lymphatic masses combined with nitrogen mustard used systemically and sometimes locally (i.e., in the pleural cavity). Chlorambucil is often beneficial in the treatment of Hodgkin's disease. Actinomycin-D appears promising in combination with irradiation. ACTH and adrenal hormones may cause temporary regression of lymphosarcoma and may at times reduce the hemorrhagic tendency in patients with bone marrow involvement.

Medical treatment combined with irradiation may prolong life to a median survival of 7.5 months with lymphosarcoma and 40 months with Hodgkin's disease. Some patients with localized Hodgkin's disease may survive as long as 25 years after surgical removal and irradiation therapy.

WILMS'S TUMOR

Behavior and General Characteristics

This renal tumor is composed of embryonic renal tissue. Most frequently it appears as a mass in the child's abdomen. It may be on either side of the abdomen, and occasionally is bilateral. Sometimes there will be fever. This generally means that there is necrotic tissue or hemorrhage within the tumor. Occasionally the first sign will be blood in the urine. This is a most unfavorable prognostic sign, since it means that the neoplastic tissue has extended outside its capsule. Wilms's tumors may grow rapidly, but the size alone bears no relationship to prognosis. Some of the largest tumors may be curable.

Other conditions which must be considered in the presence of a

large abdominal mass in a child are neuroblastoma and hydronephrosis. When a patient with such a mass is seen, an intravenous pyelogram must be made without delay. When a Wilms's tumor is small, the urogram may show nothing, but typically there is increase in the size of the kidney with distortion and even destruction of the calyces, infundibula and pelvis (Fig. 111). Occasional irregular calcification or ossification is different than a speckled neuroblastoma. Chest films

Fig. 111. Wilms's tumor. Large right renal mass with deformity of the calyces and renal pelvis.

are also indicated, since Wilms's tumor metastases are most frequently pulmonary in contrast to the osseous metastases of neuroblastoma. A hydronephrosis large enough to present a mass will often not concentrate sufficiently to show on the films. Occasionally an upright film a few hours after the injection may show opaque material dependent in the calyces or pelvis.

The tumor is generally encapsulated, yet it may spread locally. It usually metastasizes early to the lungs and may extend directly into the renal vein and inferior vena cava. If there is no obvious evidence

of metastasis, the prognosis, with proper treatment, varies inversely with age. The younger the patient, the better is the chance for cure.

Treatment

The best results in the treatment of Wilms's tumor have been obtained by a combination of surgery and irradiation. Ladd,[8] Gross[5] and Swenson[11] all feel that surgical removal of the tumor should be performed as soon as the diagnosis is made, followed immediately by irradiation. The over-all record reported by Gross[5] from the Boston Children's Hospital is the best yet seen. During a recent seven-year period they have had a 47.3 per cent cure for all ages, with 80 per cent of those 12 months of age or less. We therefore follow their methods. On the other hand, Harvey[7] states in his review of 250 cases that a cure rate of 32 per cent has been obtained by using irradiation before as well as after surgery.

The surgical treatment most widely used at present for Wilms's tumor was described by Ladd and White[8] in 1941. The essential of their approach is transabdominal excision of the mass as soon as the diagnosis is made. By this means the renal vessels may be interrupted as the first step to prevent tumor cells from being dislodged into the general blood stream. Because of the rapid growth, early metastasis, and sometimes breakdown of the tissues, it is important to perform the operation immediately. In their plan of treatment irradiation to the operative site is begun within a few hours after the completion of operation.

During x-ray treatment there is danger that a dose sufficient to cause fatal radiation nephritis may be delivered to the remaining kidney. Treatment should be directed to the tumor bed and to the adjacent lymph nodes in the periaortic area. The dose level should be in the range of 2500 to 3000 roentgens in air, remembering to spare the normal kidney as much as possible.

Medical treatment of Wilms's tumor is unsatisfactory. Nitrogen mustard has little effect. Actinomycin-D has sometimes helped cause regression of pulmonary metastasis. The latter, in combination with irradiation, is used by us for metastases of this neoplasm.

BONE TUMORS

Primary malignant skeletal tumors show a predilection for the ages between 5 and 25, with the peak incidence in late adolescence. The types usually encountered in children and adolescents are chondrosarcoma and osteogenic sarcoma (80 per cent), Ewing's tumor (15 per cent), reticulum cell sarcoma and rare types (5 per cent). Malig-

nant degeneration of benign osteochondromas or giant cell tumors does not occur in children.

The behavior of these tumors is consistent, the usual symptoms being pain, sometimes coincident with trauma. Sarcomas arise usually in the metaphyses or shafts of long bones and, if advanced, may show dusky rubor and increased venous pattern with elevated skin temperature and tenderness. In Ewing's sarcoma, fever and leukocytosis may mistakenly suggest osteomyelitis.

Radiologic Features

Malignant bone tumors show both destruction and proliferation. Elevation and lamellation of the periosteum are frequent. Cortical destruction and a contiguous streaked soft tissue mass suggest malignancy (Fig. 112). The differential diagnosis of lesions producing lytic destruction includes benign bone cyst, acute osteomyelitis and Brodie's abscess, tuberculosis and syphilis. Other lytic lesions to be considered are metastatic malignancies such as neuroblastoma, and benign lesions of dis-

Fig. 112. Osteogenic sarcoma. Bone production with little destruction. Soft tissue spicules confirm malignancy.

seminated type such as eosinophilic granuloma. Lesions producing new bone which must be distinguished particularly from osteogenic sarcoma include osteomyelitis, osteoid osteoma, benign osteochondroma and osteoma. Ewing's tumor imitates benign chondroblastoma and reticulosis. Extreme callus formation, seen in infancy after fracture, has been misdiagnosed as malignancy and thereby caused tragic amputation of an extremity. Infantile cortical hyperostosis should not be confused.

Treatment

Present treatment resolves itself to a choice between, or combination of, surgical eradication, locally or by amputation, and irradiation. Despite the inescapably poor prognosis of these tumors, there are proved cases of cure of all the common types. Reticulum cell sarcoma may yield as much as 50 per cent survival. Coley and Harrold[4] report on 252 cases of osteogenic sarcoma with 59 survivals for 5 or more years. They conclude: "It is hoped to dispel the attitude that this disease is uniformly fatal."

All cases should be handled methodically. *First*, clinical investigation to rule out metabolic and infectious disease, and radiographic survey for metastases or multiple lesions. *Second*, adequate biopsy after conferring with radiologist and pathologist as to site and method. *Third*, decision on surgery or radiation, or both. The tendency at present is to favor initial heavy radiation followed by high amputation in chondrosarcomas and osteogenic sarcomas of extremities. In slow-growing, low-grade lesions radical local excision may be warranted. Radiation alone is favored in the treatment of reticulum cell sarcoma, which is radiosensitive. Equal division of opinion exists on these two courses of treatment for Ewing's sarcoma.

In the presence of metastases, palliative resection, amputation or irradiation may still be indicated to relieve pain.

If irradiation is to be used for cure, the dose should be carried to 3500 to 4000 roentgens in air, even though there is the possibility of radiation damage to skin or bone. When treatment is for palliation of bone or joint pain, the dose should be kept low. If no pain relief is obtained by 1200 to 1500 roentgens, the addition of more will not change the picture.

SOFT TISSUE SARCOMAS

Behavior and General Characteristics

Sarcomas of the soft tissues comprise 6 per cent of our series. They include fibrosarcoma, rhabdomyosarcoma, liposarcoma, malignant neurilemmoma and synovioma. Because some benign-appearing tumors may

have foci of sarcomatous change, they should always be studied by means of multiple histologic sections. Some fibrosarcomas of the extremities are nonmetastasizing, although locally infiltrative and prone to recurrence.

The reticulum cell sarcoma found occasionally deep in the soft tissues involving muscle sheath may or may not be related to the reticulum cell sarcoma arising in the hematopoietic system. It behaves, however, as a fully malignant metastasizing neoplasm.

One of the most frequent of the childhood sarcomas is the rhabdomyosarcoma which occurs in the genitourinary tract, diaphragm, head, neck and other areas. This tumor spreads by direct extension and through the lymphatics.

Treatment

Since the soft tissue sarcomas are seen in many instances prior to lymphatic or vascular spread, a determined effort should be made to remove them through wide local excision. Many may seem to be encapsulated and yet histologically will be found to have finger-like extension outside the apparent area of tumor. When one group of lymph nodes drains an involved area, this group should be removed.

The rhabdomyosarcoma, especially, should be dealt with by wide local removal. When encountered externally, as in the head and neck, this offers some chance of success. When the tumor is internal, the prognosis is less good. Fibrosarcomas of the extremities should be excised with a good margin, without amputation.

Pack[9] has observed a 5-year cure rate of approximately 50 per cent among childhood soft tissue sarcomas.

These tumors vary in their sensitivity to irradiation. Occasionally in children one with lipoid or myxoid elements predominating will show response, and in those instances the total dose should be carried to levels of 1500 to 3000 roentgens to the tumor.

This group of malignancies generally responds poorly to nitrogen mustard and similar polyfunctional alkylating agents. The x-ray potentiating effects of actinomycin-D may cause regression of small lesions, but not of large rapidly growing masses.

CARCINOMA

Behavior and General Characteristics

In our study of 408 cases of malignancy we found 18 carcinomas. The average age was 9 years, while 8 children were 11 years or over. This means that a large proportion of the group was approaching or had passed puberty.

Our largest single group, of which there were five, was of the thyroid. *Thyroid* carcinoma frequently presents as an enlargement of cervical lymph nodes filled with metastatic tumor. It may also metastasize to lung and bone. Histologically, the tumor is of follicular adenocarcinoma variety, showing varying densities of colloid material. This neoplasm is characterized in some instances by a most unusual longevity even in the presence of metastases. Some patients may live comfortably 10 to 15 years after metastases have appeared in the lungs. (See also p. 1019.)

In every type of carcinoma which we have seen the clinical characteristics and behavior could be said to be about the same as those of a similar tumor in the adult age group. Examination of our list of carcinomas (Table 4), however, will demonstrate the generally accepted

TABLE 4. *Carcinomas Seen among 408 Cases of Malignancy in Infants and Children**

	AGE IN YEARS
Adenocarcinoma of kidney	13
Clear cell carcinoma of kidney	8
Adenocarcinoma of thyroid	14
" " "	13
" " "	9
" " "	8
" " "	3
Mucoepidermoid carcinoma of palate	11
Carcinoma of nasal mucous membrane	14
Squamous cell carcinoma of lip	12
Mixed tumor of parotid	14
Sebaceous carcinoma, preauricular	13
Carcinoma of salivary glands	5
Carcinoma of liver	1
" " "	1
Carcinoma of adrenal cortex	8
" " " "	4
Embryonic carcinoma of testis	2
Average age	9

*From the Children's Orthopedic Hospital and the Tumor Institute of the Swedish Hospital, Seattle,[1] 1949–1958.

fact that the common carcinomas of the adult age group are seldom or never seen before puberty. Absent from our list are carcinomas of the skin, gastrointestinal tract, breast, lung, uterus and bladder.

The feature distinguishing *adrenocortical* carcinoma from the benign corticoadenoma is invasion of the capsule by the tumor. This must be determined by histologic examination. Early metastasis to lymph nodes and lung is the rule in embryonal carcinoma of the *testis*. Carcinoma of the *liver* may show aggregations of atypical hepatic cells or sometimes an undifferentiated embryonic histologic picture. These tumors may remain localized to the liver or spread freely by metastasis.

Mucoepidermoid tumors are generally found in the parotid gland, although a few are seen in the submaxillary salivary glands. These should be regarded as malignant, and wide excision of the local tumor should be carried out. A certain proportion of these tumors present a low-grade type of malignancy which may be mistaken for a completely benign tumor. Recurrence may develop locally, and metastases tend to occur in bone, lung, brain and subcutaneous tissue.

Treatment

The surgical treatment, irradiation and chemotherapy of carcinomas should be the same as that for the same type of tumor in the older age group. No drug is known to be consistently effective. A trial of nitrogen mustard and similar agents, and even actinomycin-D, may be worth while in patients with metastatic lesions. Radioactive iodine (I^{131}) in thyroid carcinoma may be palliative. Testosterone has caused regression of mucoepidermoid carcinomas in a few preadolescent children.

TERATOMA

Behavior and General Characteristics

Teratomas contain elements from mesoderm, entoderm and ectoderm, and they generally display some degree of progressive uncoordinated growth. They may actually be thought of as an abortive attempt at the formation of a twin. In a study of 23 teratomas of the sacrococcygeal region, Gross, Clatworthy and Meeker[6] in 1951 found that there was a close family history of "twinning" in half of these cases. *Although these tumors are at first benign, they have the potentiality of becoming malignant.*

In our series of 15 teratomas, 5 were in the testis, 5 in the sacrococcygeal area, 3 in the mediastinum, 1 was retroperitoneal, and 1 was in the ovary. These tumors may occasionally achieve enormous proportions compared with the size of the child. This is particularly true of the sacrococcygeal teratoma which may be present at birth. This particular type of tumor will be found to have its origin from the anterior portion of the sacrum and coccyx. Teratomas within the chest usually appear within the anterior mediastinum and are similarly well encapsulated in their early stages (Fig. 113). Such tumors may become infected or may rupture into the pleural cavity or pericardium. When malignant degeneration does take place, it is generally of but one of the cellular components of the teratoma.

For x-ray identification of these lesions oblique films are of great aid in searching for calcification, ossification and teeth elements in midline tumors in any location of the body.

Fig. 113. Teratoma. Anterior mediastinal tumor arising from the midline and extending to the left. Ossification strongly suggests teratoma.

Treatment

Because of the malignant potential of these tumors, surgical treatment should be uniformly directed toward complete removal as soon as the diagnosis is made. In the instance of the sacrococcygeal teratoma, the mass should be removed through an approach posterior to the anus and with removal of the coccyx. It should then be shelled gently and accurately from the pelvis and the anterior portion of the sacrum in order not to disturb the nerve supply to the bladder and rectum.

When malignant extension and metastasis have already taken place, palliative irradiation should be used. The success in this type of therapy will depend on which type of cell within the teratoma has undergone malignant change.

Chemotherapy may also be used when metastasis or spread has appeared. The type of drug to be used will also depend on the cellular structure of the neoplasm.

GENERAL MEDICAL MANAGEMENT OF THE APPARENTLY INCURABLE CHILD

Medical management of a child with apparently incurable malignancy can be extremely heart-rending to the physician, and yet, when it is administered skillfully and sympathetically, it can give great comfort to the patient and to his family. Beyond measures to control the disease, the child's comfort should be uppermost in the physician's mind. Tranquilizing drugs and narcotics should be given when necessary. A blood transfusion may help the little patient in the later stages of disease when there has been bleeding or when bone marrow failure has appeared. The use of adrenal steroids may improve well-being when all other therapeutic measures have failed.

After x-ray therapy it is first of all important to maintain the child's electrolyte balance and protein levels. In order to do this one of the antiemetic drugs may be necessary for about a week if the child starts vomiting. If this is not entirely effective, intravenous fluids may be used temporarily. Beyond the acute stages of vomiting the child should be given adequate amounts of vitamins, particularly B complex and vitamin C. He should also be given iron.

The various chemotherapeutic agents are directed toward destruction of, or retardation of growth of, the tumor mass itself. They may

TABLE 5. *Chemotherapeutic Agents Frequently Used in Pediatric Malignancies*

AGENT	ROUTE	USUAL DOSE	INITIAL TOXICITY
Polyfunctional alkylating agents: HN2 (nitrogen mustard, Mustargen)	I.V.	0.4 mg./kg./course	Nausea and vomiting
CB 1348 (Chlorambucil)	Oral	2–12 mg./day	Occasional nausea and vomiting
TEM (triethylene-melamine)	Oral	2.5–5 mg./day; not more than 10 mg./week	Occasional nausea
Amethopterin (Methotrexate)	Oral	1.25–5 mg./day (single dose/day)	Mouth ulcers
Adrenal hormones: ACTH	I.V.	10–25 mg./day	Fluid retention
Cortisone	Oral	50–100 mg./day	Hypokalemia, hypertension, convulsions
Hydrocortisone (Solu-Cortef)	I.V.	50–100 mg./day	
Prednisone	Oral or I.V.	20–150 mg./day	
Actinomycin D	Oral	2–10 mg./day	Nausea, vomiting, mouth ulcers
	I.V.	75 γ/kg./course in 4 or 5 doses	

thus lengthen the life span considerably and may also increase the patient's comfort. The use of the various chemotherapeutic drugs has been outlined briefly in each section and also in Table 5. Before such an agent is used, one should always make certain that there is a normal blood urea nitrogen and a normal serum uric acid level. During treatment with such agents it is important to supply adequate fluid intake.

SUMMARY

Malignancy is now second only to accidents as the cause of death in children. The types of malignancy seen in infancy and childhood are generally unique to this age group. Although 90 per cent of the malignancies in the adult age range are carcinomas, this type comprises less than 5 per cent of neoplastic disease seen before puberty.

Following leukemia and brain tumors, the common types of malignancies encountered in children are, in order of frequency, (1) neuroblastomas, (2) lymphoid tumors, (3) Wilms's tumors, (4) bone tumors, (5) soft tissue sarcomas, (6) carcinomas, (7) teratomas.

A large proportion of these patients can be cured by the proper use of surgery, x-ray therapy and chemotherapy. The methods to be used vary with the type of malignancy encountered.

The young patient apparently incurable by surgery may frequently be given an increase in life span by the intelligent use of irradiation and chemotherapeutic drugs. These agents together with other measures will, as a rule, greatly increase their comfort and well-being.

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NEUROSURGERY IN THE PREVENTION OF EXOGENOUS MENTAL RETARDATION

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The past decade has witnessed an emphasis upon the emotional and the psychologic aspects of pediatrics to a remarkable degree. Though initially such emphasis may have been necessary, the pendulum of pediatric thought has swung so far toward this point of view that the more direct and frequently more obvious organic difficulties are often overlooked. It is now pertinent to recall certain basic principles and to reaffirm the fact that disease of the brain and the mind are frequently related and that the capacity of a normal brain for intellectual growth may be altered by organic entities, some of which lend themselves particularly to surgical therapy.

The recognition of such surgical conditions early in infancy, and particularly the need for early therapy so as to prevent irreversible brain damage and subsequent clinical retardation, is the burden of these pages.

The common neurosurgical conditions to be discussed—subdural hematoma, craniostenosis and hydrocephalus—have several basic similarities.

1. Untreated, all will result in progressive brain compression with secondary cerebral circulatory depression.

2. Early in the clinical course there may be preservation of the patient affect despite motor retardation.

3. Finally, early surgical decompression, particularly in the first few months of life, will usually result in significantly improved cerebral function.

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THE SUBDURAL HEMATOMA OF INFANCY

The subdural hematoma is both a common and a protean lesion during infancy. Despite frequent references in the literature, it is all too often overlooked. Although treatment may be ultimately a surgeon's responsibility, the decision as to the need for investigation and the early diagnosis rest with the pediatrician or general practitioner. The dictum "to think of a subdural is to find it" cannot be overemphasized, largely because there is no clinical picture *specifically* diagnostic of the lesion, nor is there likely to be a stereotyped history.

Etiology

Neonatal trauma or direct injury during delivery may result in subdural bleeding, but most commonly the lesion has its inception during labor—a time when neither mother nor obstetrician can materially alter the course of events.

Avitaminosis and bleeding diathesis are often mentioned as possible causes, but are rarely found. Of more recent appreciation, the clinical recognition of subdural collections secondary to leptomeningitis has become increasingly significant. As an etiologic factor in our series of approximately 350 patients, meningeal infection ranks second in historical frequency only to trauma during a difficult and protracted labor.

Clinical Features

The symptoms and signs of this disease are nonspecific. Irritability, vomiting, convulsions, along with full fontanel, enlarged head, fever, anemia, abnormal fundi, are found also in many other entities. X-rays of the skull are of questionable value, although they occasionally show a fracture or slight separation of sutures. The so-called subdural skull (Fig. 114), brachycephaly associated with a wide biparietal diameter, is often noted, but again is not specific. In fact, several children with borderline clinical microcephaly proved to have chronic subdural collections and improved remarkably after decompression. In short, so misleading may the clinical picture become that it is now the rule at The Children's Hospital of Philadelphia to look for a subdural collection as a prime cause until proved otherwise in all infants who demonstrate any aberration from the norm in development.

Diagnosis

During the course of the clinical examination transillumination may prove useful, but the subdural aspiration still affords the most certain

A B

Fig. 114. Unretouched postoperative photographs of a child with bilateral subdurals, demonstrating the brachycephaly described.

diagnosis, short of direct visualization. The taps are technically uncomplicated and with experience become diagnostically precise. Maintenance of sterile technique is essential. The use of a short needle with stylet and placement of the puncture site sufficiently lateral to the midline sagittal sinus and the zone of origin of the bridging veins will preclude the remote possibility of secondary hemorrhage.

Occasionally, air study may demonstrate a subdural hematoma which has escaped detection by aspiration. Consequently, pneumoencephalography should be utilized in the face of a negative subdural tap whenever the index of suspicion is sufficiently high.

Pathology

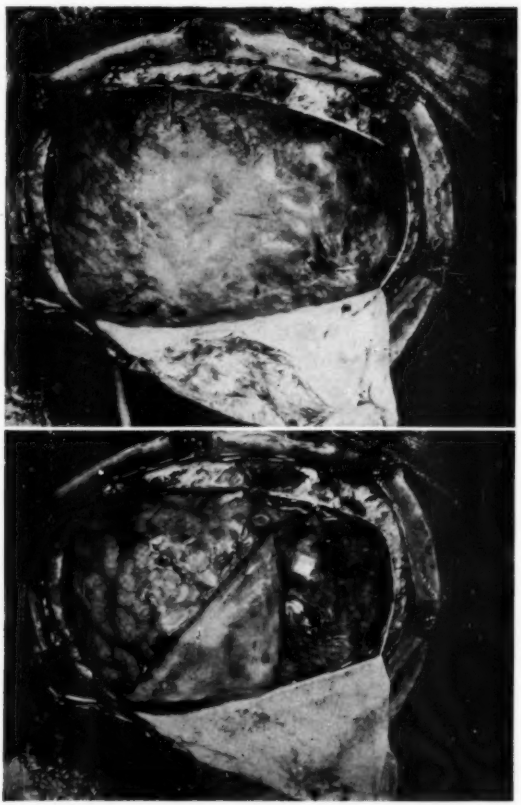
In general, subdural hematomas in infants will involve the anterior and superior portions of the cerebral hemispheric surface. This observation suggests that rupture of one or more bridging veins is largely responsible for the initial bleeding.

In our infant series more than 70 per cent of the subdural hygromas occurred bilaterally, although they were not always equally extensive.

The acute phase of the hematoma is the simple accumulation of varying amounts of whole blood within the subdural space. Within a matter of days (7 to 12 days average) in those infants in whom the blood is not resorbed, a proliferation of fibroblasts takes place, primarily from the under surface of the dura. This may be surprisingly thick and tends to become vascularized and relatively inelastic. When

the reactive membrane is joined peripherally by a similar but usually thinner membrane originating from the arachnoid, the original hematoma literally becomes a sac containing whole blood elements. The sac wall acts as a dialyzing membrane when in contact with the contiguous pathologically altered arachnoid. In time, through osmosis, water derived from the relatively low protein cerebrospinal fluid enters the fibrous sac to reduce progressively the protein content of the blood within. The subdural fluid may indicate the age of the lesion by virtue of its color and protein content. The more recent chronic subdural fluid collection with its well formed membranes and highly protein fluid is often a dark red brown. In time the fluid may become orange

A



B

Fig. 115. The more typical subdural hygroma. A, The dura open and membranes exposed. B, Beginning removal of membranes.

or xanthochromic. In chronic lesions the fluid may be only slightly yellow, although the protein concentration may still be in the order of 800 to 1000 mg. per 100 ml. Such chronic subdural collections are known as *hygromas* and are accompanied by well organized neomembranes and highly protein fluid (Fig. 115).

In contrast, the term subdural *hydroma* has come to mean a subdural fluid collection regardless of etiology with a low protein content (rarely as high as 200 gm.) and with poorly defined fibrinous membranes or none at all.

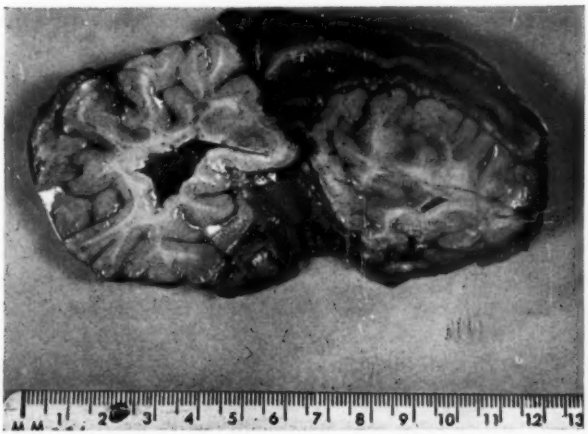


Fig. 116. The unfortunate result of an undiagnosed subdural hygroma—an incidental finding at autopsy in a 12-month-old retarded child who died because of severe bilateral pneumonitis.

Clinical Course

Cerebral atrophy is the uniform result of the untreated chronic subdural hygroma (Fig. 116). If the delay in cerebral decompression is unduly prolonged, permanent brain damage with secondary mental and motor defect occurs. An arbitrary top limit of nine months of age prior to decompression correlates well in our series with a satisfactory clinical result.

Surgical Treatment

Despite a variety of therapeutic attempts to avoid craniectomy in these youngsters, our series of patients demonstrates without question that whenever trephination and biopsy demonstrate a true fibroblastic neomembrane (Fig. 117), an ultimately satisfactory result is obtained only by removal of both the subdural fluid collection and the patho-

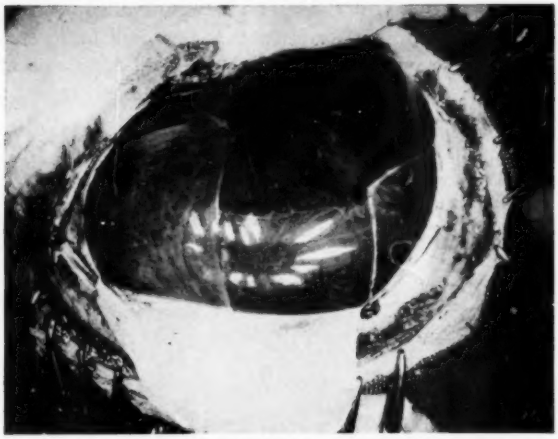


Fig. 117. Operative view of a massive subdural hematoma, demonstrating thin but well formed membranes 7 days after a traumatic incident.

logic membranes. Only in those patients in whom *fibrinous pseudomembranes* are found at trephination have we observed a satisfactory result without removal of the membranes following the evacuation of the subdural fluid.

The therapeutic routine utilized in each patient will depend both on the chronicity and extent of the lesion and on the age and condition of the patient. Because of generally earlier diagnosis, the severe debility noted in the subdural patient of 10 years ago is seen less frequently today. The consequent better patient risk makes it possible to reduce the initial preoperative phase of repeated subdural aspiration to a few days rather than weeks. However, it is still necessary to use caution in tapping in order to prevent too rapid decompression and subsequent shock.

Although repeated needle aspiration may apparently dry up the subdural collection, the second therapeutic phase, that of trephination, is obligatory, particularly if the original subdural fluid contained a high protein concentration (800 to 1000 mg. per 100 ml.). The trephine openings should be bilateral, and located over the anterior and superior portion of the cerebral convexity. Membranes, if present, should be biopsied.

The presence of typical neovascular formation and fibrosis in the specimen will make craniotomy, the third and final therapeutic phase, a necessity.

Great strides in anesthesia technique coupled with the use of adequate amounts of whole blood early in the course of the craniotomy procedure have reduced mortality to the vanishing point.

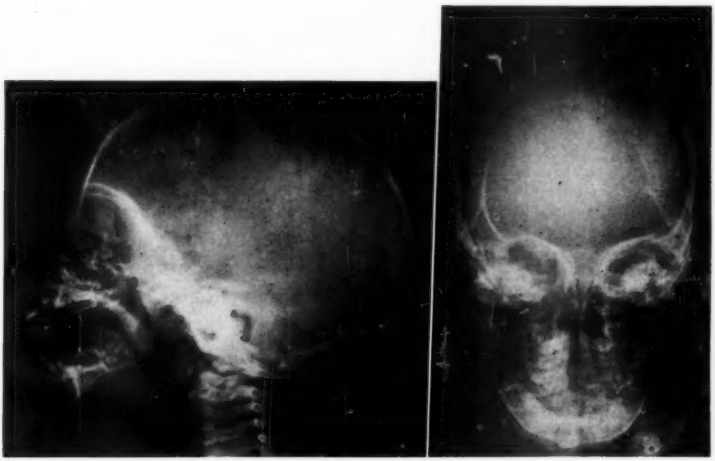
RESULTS. Complications incident to the procedure are uncommon. Postoperative convulsions occurred in less than 10 per cent of our most recent 250 patients. Reoperation because of spontaneous recurrence is also extremely rare, having occurred in only three patients in our total series.

It is important to re-emphasize that operation carried out prior to severe cerebral atrophy gives the best results.

In the years 1948–1953 some 65 per cent of our patients demonstrated a normal developmental pattern. In the last 5 years more than 80 per cent of the operated group have developed normally. These encouraging results are an indication of the increasing awareness of the subdural hematoma among clinicians and particularly of the salutary effect of earlier diagnosis and therapy.

CRANIOSTENOSIS

Of the three entities under discussion, craniostenosis, or more correctly craniosynostosis, is probably the least understood from the standpoint of specific etiology. Because of the frequency of its occurrence in successive generations and in siblings, it has been postulated that an inherent germ plasm defect must be present. The more common occurrence of the process in the male and the not infrequent association with syndactylism and congenital heart disease would reinforce this



A B

Fig. 118. Lateral (*A*) and posteroanterior (*B*) views of a patient with borderline microcephaly and primary cerebral dysgenesis referred to us as a craniostenotic. As the x-rays demonstrate, however, no premature closure of the sutures is present, and operation would have had no beneficial effect.

Fig. 119. Clinical appearance of the scaphocephalic infant with premature closure of the sagittal suture.

hypothesis. Other etiologies from prenatal trauma to endocrinopathy have been suggested, but none has met the test of time. In at least two instances in our experience, increased vitamin intake by a misguided parent has been associated with premature closure of the sagittal suture. In no instance in our experience has the pathologist demonstrated an abnormality of periosteum or skull.

Despite the uncertainty as to cause, the syndrome of brain compression secondary to craniosynostosis was treated surgically as early as the end of the last century. Unfortunately, surgical therapy fell into disrepute because of the clinical confusion between microcephaly and craniostenosis.

It should be remembered that expansion of the skull is entirely dependent on growth of the underlying brain. During the first year of life the brain more than doubles its weight, and between 50 and 60 per cent of the total circumferential increase in the cranial vault occurs during the same interval. In microcephaly the sutures do not close prematurely. Consequently, the serious developmental defect in these children is not the result of brain compression incident to premature suture closure; rather, the small cranial vault results from inadequate expansion of a primarily defective brain.

At birth the cranial bones of the normal infant are separated. Fibrous union may occur by the sixth month. Calcification, however, is frequently delayed until the fifth decade or later. Consequently, should calcification and therefore closure of an important cranial suture occur in utero or shortly after birth, there will be inevitable secondary brain compession at a time when the total brain mass is attempting to expand rapidly.

All too commonly there is inadequate appreciation of the fact that irreparable brain damage may occur before the onset of the classic signs and symptoms of increased intracranial pressure. As early as 1927 Farber pointed out the need for early operation, but it was not until 1948 that the need for early operation was sufficiently re-emphasized.

It is the unqualified feeling of The Children's Hospital group that early recognition and decompressive surgery in the first few months of life will not only improve the infant's grotesque appearance, but also, of even greater importance, will prevent the occurrence of mental retardation, convulsive seizures, behavioral aberrations and visual and

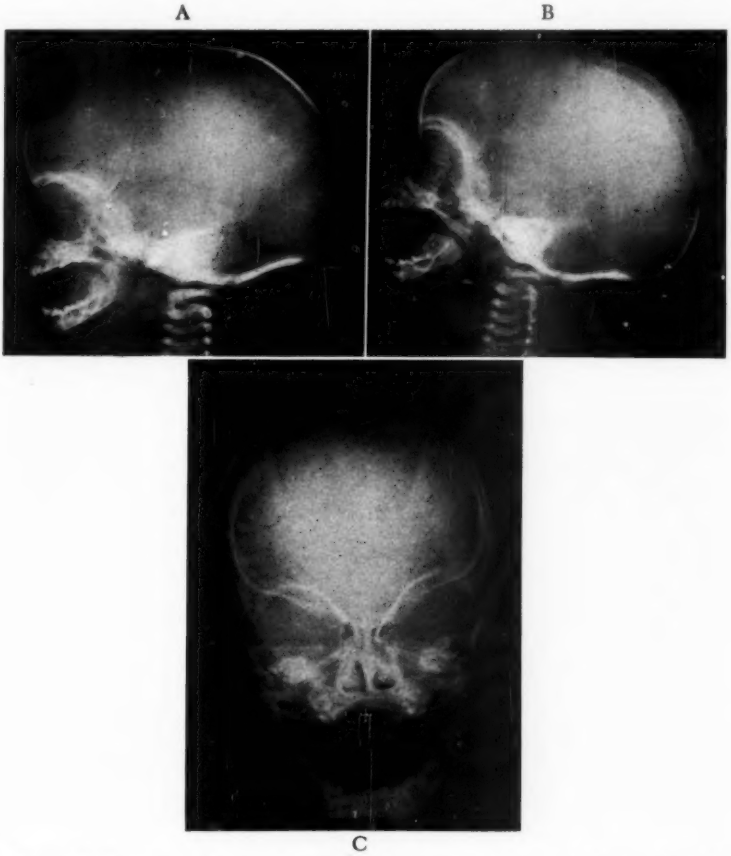
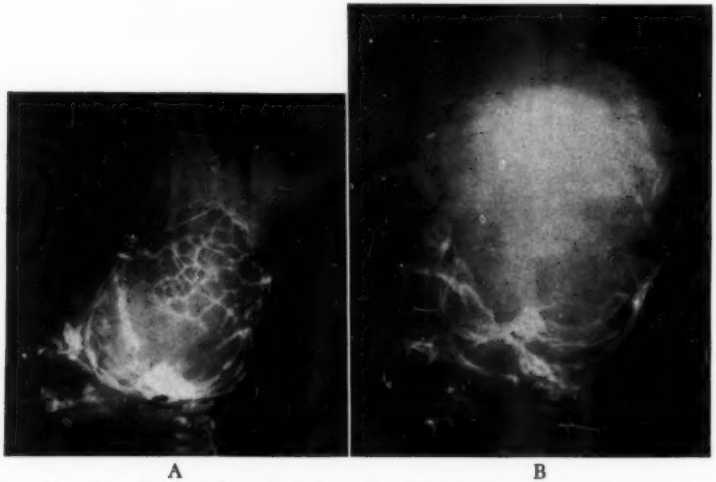


Fig. 120. Premature closure of the sagittal suture, and its surgical correction. *A*, Typical preoperative lateral view. *B*, Immediate postoperative roentgenogram, demonstrating removal of the sagittal suture. *C*, The same child 1 year after operation. Note the improved contour of the skull.

A B

Fig. 121. Preoperative (*A*) and postoperative (*B*) clinical appearance of a patient with extreme degree of craniosynostosis involving all the sutures.



A B

Fig. 122. Preoperative (*A*) and postoperative (*B*) roentgenograms of the patient shown in Figure 121.

auditory defects so often noted singly or in combination in the neglected patient.

Diagnosis

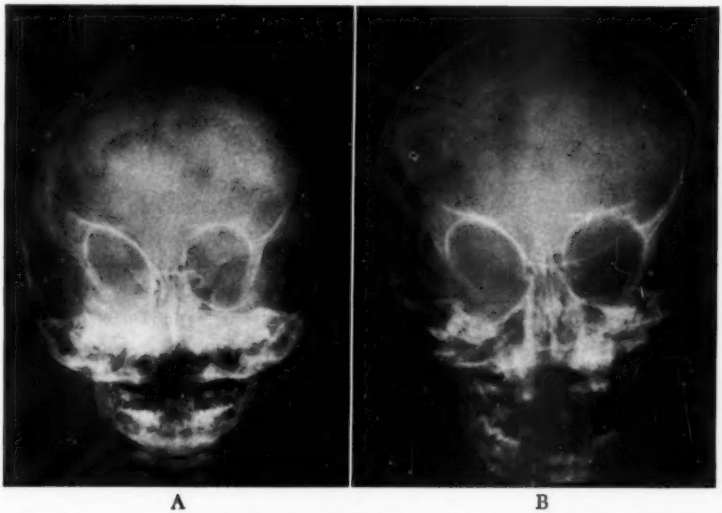
The clinical diagnosis of craniostenosis is made largely on the basis of a misshapen skull early in the course of the infant's life. Skull distortion thought to be secondary to a difficult birth should always be suspect if the abnormal appearance persists. In all cases, however, the precise diagnosis depends on x-ray verification.

Of particular importance clinically and worthy of illustration are those cases having (1) premature closure of the sagittal suture, (2) premature closure of all sutures, (3) premature closure of both coronal sutures, (4) premature closure of a single coronal suture, and (5) premature closure of the metopic suture.

Therapy

Rather than describe the surgical therapy in detail, the basic surgical principles will be emphasized:

1. In all cases an attempt is made to remove surgically the calcified suture or to provide substitute parallel artificial sutures.



A B

Fig. 123. *A*, Extreme distortion and angulation of the lateral and superior margins of the orbit on the affected side. *B*, The rounding and general improvement in the state of the orbit following decompression. (We prefer to carry out the decompression of the unilateral coronal suture even earlier than in this case. In this patient earlier operation would have brought about a better result.)

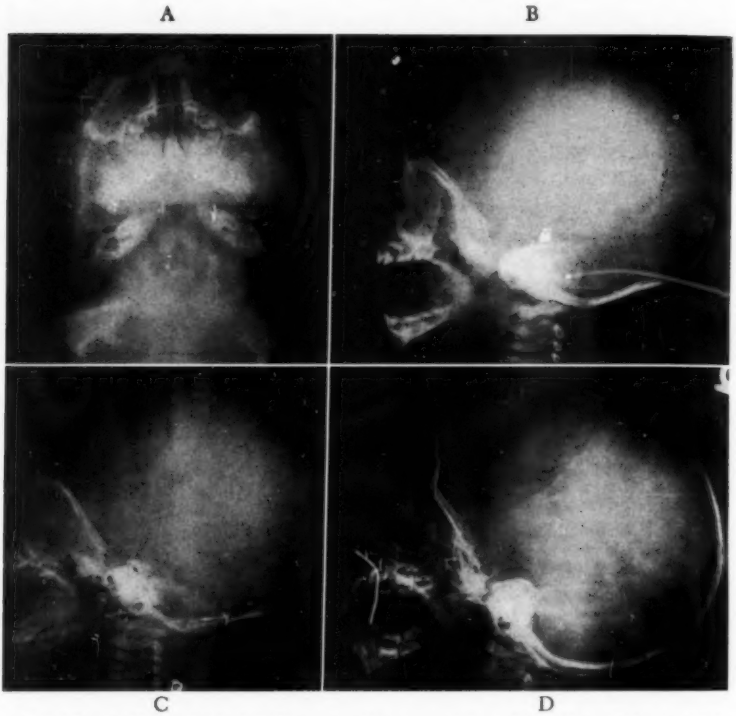


Fig. 124. A, Tangential view demonstrating premature calcification of the metopic suture. *B*, The same patient, preoperative lateral view. *C*, The same patient immediately after operation, demonstrating the type of decompression obtained. *D*, The same patient, 18 months later, demonstrating the increase in size of the anterior fossa anteriorly and also the improved contour of the forehead.

2. Occasionally, if flattening of the skull contour with constriction of an intracranial quadrant has occurred, morcellation of the affected area may prove useful. This will result in a more satisfactory cosmetic effect, but particularly will permit of greater brain growth.

3. An attempt is made whenever possible to prevent too rapid regrowth of bone by lining the edges of the surgical decompression with suitable material. In recent years polyethylene, Kel F, and silicone sheeting have all been used and have proved satisfactory.

Results. Complications incident to surgery are most unusual. Mortality is nonexistent, and the postoperative course is generally uneventful. Reoperation, however, may be necessary in some instances. In not a few cases, when the operation is carried out in the first weeks of life, reoperation may be expected at least once by age three and one-half years. This is about the time when the brain has developed the

bulk of its functional organization and size. For this reason postoperative follow-up is thorough, and occasional x-ray check-up to age four years is imperative. In our series relatively few sagittal premature closures need reoperation. As high as 30 per cent of the bilateral coronal suture patients have had at least one reoperation by age 3 years.

In the last several years, largely because of the insistence of our pediatric colleagues, an occasional older child (6 to 12 years) with previously unrecognized craniostenosis and severe emotional difficulties has been significantly improved by surgical decompression.

Summary

The diagnosis of craniosynostosis may frequently be obvious at birth or early in the neonatal period. An unusual skull formation should suggest the possibility, and final verification of the diagnosis will depend on an x-ray examination.

The differential diagnosis between craniostenosis and microcephaly should be appreciated. The latter type of patient is usually grossly defective, and the x-rays fail to demonstrate premature suture closure.

The last word has not been spoken about craniostenosis, but certainly we have learned that surgical decompression early in the first year of life, prior to the onset of the signs and symptoms of increased intracranial pressure, will result in a more normal child developmentally.

HYDROCEPHALUS

Hydrocephalus is at once the most common and among the most stimulating pathologic states with which the pediatric neurosurgeon must deal. A recognized entity since antiquity, it has classically been considered incompatible with normal intellectual and motor development, and uniquely devoid of therapeutic success. Fortunately, this uncompromising picture of failure has been altered in the last decade. Today the onset of hydrocephalus with progressive distention of the cranial vault is considered far from hopeless.

The Cerebrospinal Fluid Circulation

A rudimentary understanding of the circulation of the cerebrospinal fluid is basic to any discussion of hydrocephalus. The early confusion brought about by use of the newer investigative techniques, particularly the application of radioactive isotopes, has been clarified. The original concepts of Weed and Dandy, and the importance of the

choroid plexus in the elaboration of the cerebrospinal fluid, appear to have been vindicated. Suffice it to say that there are primarily two areas of cerebrospinal fluid circulation. The first of these, entirely within the ventricular system, is best described as the intraventricular portion of the cerebrospinal fluid circulation. The second area of circulation, far more diffuse, is termed the subarachnoid portion. For practical purposes we can consider virtually all the cerebrospinal fluid as formed within the ventricles, particularly the lateral ventricles. Similarly, virtually all the cerebrospinal fluid, after traversing portions of the subarachnoid system, may be considered to be absorbed through a series of contact zones directly into the major venous sinuses.

Classification

In the normal state of health there is free communication between the ventricular and the subarachnoid cerebrospinal fluid systems. Any pathologic entity which results in a lack of communication between the two systems is said to result in a noncommunicating hydrocephalus. In contrast, should there be free communication between these systems, but a defect in absorption, either congenital or acquired, the resulting hydrocephalus is classified as the communicating type. The term "communication" therefore has reference only to the passage of cerebrospinal fluid freely between the ventricles and the subarachnoid space.

Should we omit from consideration those lesions such as tumors amenable to direct surgical attack, it becomes apparent that the development of surgical procedures which are effective in both types of hydrocephalus has materially lessened the need for the precise classification of each case.

Etiology

Generally speaking, the causes of hydrocephalus are legion, and may produce either a communicating or a noncommunicating hydrocephalus. In our own Clinic the following etiologies were found most common in order of decreasing frequency: (1) congenital communicating hydrocephalus (frequently described as idiopathic and most often apparently the result of a poorly defined defect in the absorptive mechanism), (2) noncommunicating hydrocephalus incident to leptomeningitis, (3) noncommunicating hydrocephalus secondary to neoplasm, (4) noncommunicating hydrocephalus secondary to congenital aqueductal stenosis, (5) communicating hydrocephalus secondary to meningitis, (6) noncommunicating hydrocephalus incident to subarachnoid hemorrhage with secondary basilar arachnitis.

In our series of 1586 cases the hereditary factor appeared to be of minor importance. Consequently, in response to the usual question, we minimize the dangers of the occurrence of hydrocephalus in future pregnancies unless the heredo-familial history is unequivocal.

Diagnosis

The diagnosis of hydrocephalus is rarely difficult. The infant's appearance—craniofacial disproportion, protuberant forehead, tense fontanel, distended scalp veins and, finally, a peculiar and constant stare (the so-called sunset sign)—can rarely be misinterpreted. Ready transillumination and an excessively tympanic sound to percussion further reinforce the examiner's gross impression.

The usual clinical course is one of progressive disproportion between the circumferences of the head and chest. This is not infallible, however. Occasionally an apparent cessation of cranial expansion may be grossly misleading. In such patients ventricles may become increasingly distended without a parallel increase in skull circumference.

X-rays of the skull may confirm the impression of increased intracranial pressure, but are rarely specifically diagnostic of hydrocephalus.

At The Children's Hospital of Philadelphia a great deal of significance is placed upon the result of the dye circulating technique. The routine consists of the following steps:

1. Bilateral subdural taps—if negative, the remainder of the examination is completed.
2. Bilateral ventricular puncture with determination of (*a*) brain thickness (great differences in brain thickness suggest the need for air study), and (*b*) ventricular pressure.
3. Two to 3 cc. of indigo carmine dye is placed in one ventricle, and then, with a clean syringe, the opposite ventricle is gently aspirated. The appearance of dye is usually almost immediate. Lack of dye indicates the presence of a block high in the ventricular system. In this instance ventriculography is indicated. Appearance of dye in the opposite ventricle indicates the absence of a block between the ventricles and the need for completion of the test.
4. The infant is returned to the ward, the head of the crib is elevated, and, in one hour, a lumbar puncture is carried out. Blue coloration in the cerebrospinal fluid specimen indicates that a communicating hydrocephalus is present.
5. Absence of blue dye results in a second lumbar puncture one hour later. For practical purposes, communicating hydrocephalus is considered to be present if dye is noted in satisfactory concentration in 2 hours.
6. If no dye is present, noncommunicating hydrocephalus is deemed to be present, and a cisternal puncture is carried out. Should the cisternal specimen be free of blue color, a block in the lower third ventricle, aqueduct, or high in the fourth ventricle is a distinct possibility. In such a case, air study is indicated, and frequently a combined pneumoventriculogram will prove of great value.
7. If the "cisternal" specimen contains blue dye, and the lumbar specimen does not, it is likely that the pathologic process is an arachnitis occluding the fourth ventricle outlets. This may obviate the need for air study in some cases.

The technique as described has proved more dependable than the differential simultaneous ventricle and lumbar pressure determination

and has materially reduced the number of unnecessary diagnostic air studies.

Pantopaque ventriculography utilizing the image intensifier fluoroscope has proved valuable when the bubble studies and the more complete air studies have been unable to provide an unequivocal diagnosis.

Therapy

Once the diagnosis is made, the question of therapy must be answered. This is not always easily determined, and over the years considerable controversy has ensued not only as to which type of surgical technique may be best, but also whether any attempt at therapy is justified. The latter viewpoint is frequently prejudiced by those of our colleagues who usually fall into two categories: Firstly, those who purport to show statistically that a high percentage of hydrocephalic infants will spontaneously compensate without operation; secondly, those who argue that surgical intervention is not justified because the patient who may be salvaged will be functionally defective to a considerable degree.

Our personal impression, based on considerable experience, has been that both points of view are without foundation in fact. In the first instance the cases of hydrocephalus are usually inadequately studied and rarely precisely diagnosed as to etiology, so that the results can usually be interpreted in a variety of ways, depending on one's point of view. In the second instance prolonged follow-up of many hydrocephalic children postoperatively dispels the concept of incompetence.

To my mind, we are still unable to determine clinically with absolute certainty those children who will have a satisfactory future and those who will not. Brain thickness at the time of operation is not an unquestioned indication of future cerebral potential. The final determination as to a patient's suitability for operation should remain an individual decision based on a number of factors: the apparent affect, the degree of neurologic and motor impairment, and the severity of accompanying congenital defects. All should play a role in the ultimate decision.

The variety of surgical techniques developed over the years for the treatment of hydrocephalus gives silent testimony to the difficulties encountered. These generally fall into two types: (1) attempts to decrease the amount of cerebrospinal fluid formation, (2) attempts to shunt the excess cerebrospinal fluid into a body cavity or viscus, or into the blood stream per se.

In our hands the first group has proved generally unsatisfactory. In the second group we have found generally unsatisfactory the techniques which permit of the ultimate contact of cerebrospinal fluid with the external environment. Such procedures include ventriculomastoid, and

subarachnoid or ventriculo-ureteral procedures. We have had rather pleasing long-term results with peritoneal shunts. However, over the past three and one-half years, the progressive improvement in the ventricular-vascular shunt technique utilizing the Holter valve has made this the procedure of choice in our Clinic for all varieties of hydrocephalus, communicating and noncommunicating (Figs. 125, 126). In over 400 patients we have had only 15 deaths incident to the use of the technique, and the frequency of revision has yielded to our increased understanding of the mechanical niceties of the procedure.

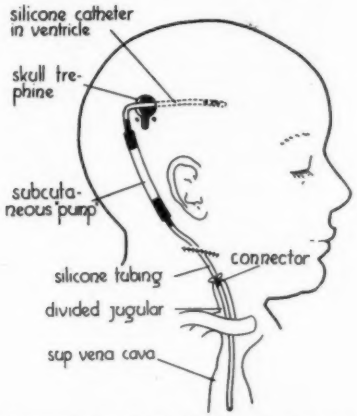


Fig. 125. Diagrammatic representation of the present ventriculo-caval technique.

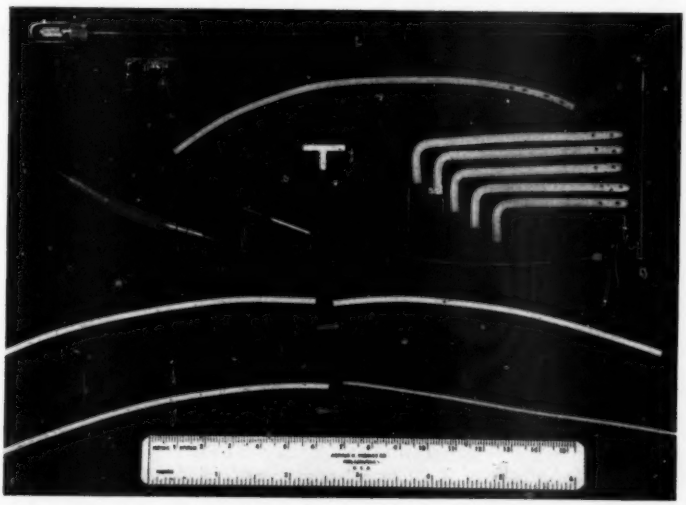


Fig. 126. The Holter valve and associated apparatus as used in the ventriculo-caval shunt.

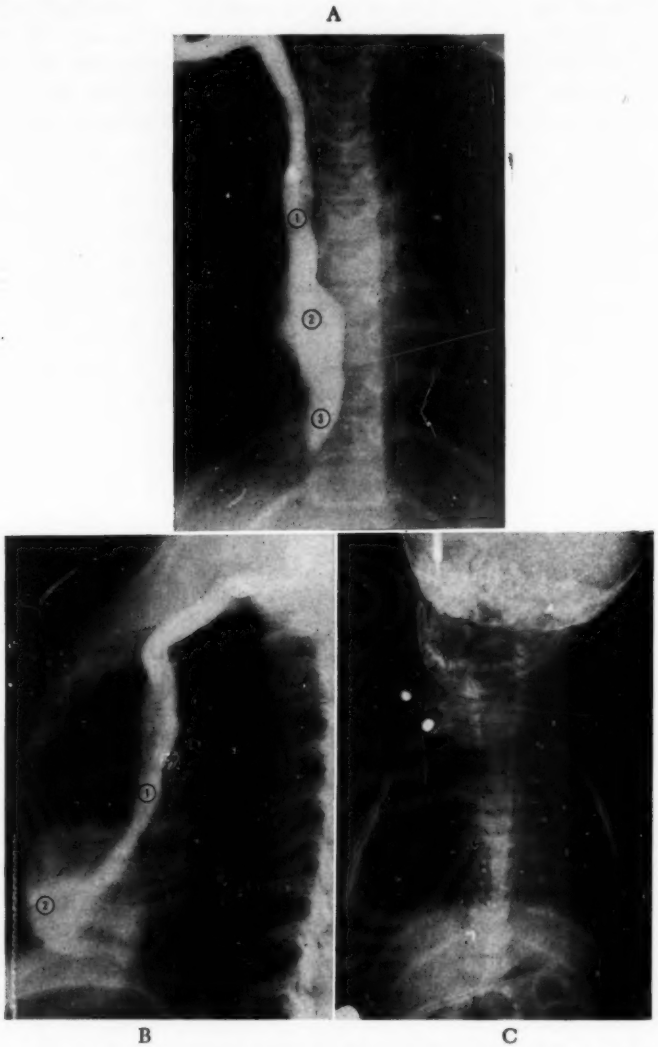
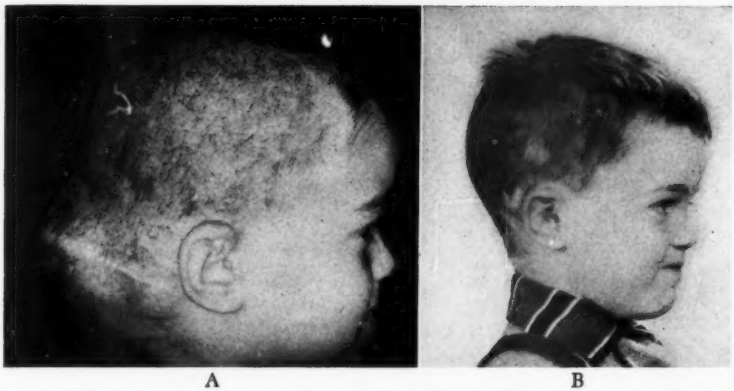


Fig. 127. *A*, Anteroposterior angiogram demonstrating (arrow) the level of the azygos vein, which in this instance coincides with the level of the carina. In the earlier shunts the end of the drainage catheter was placed a short distance below the carina. Through experience it was learned that the optimal position is at the junction of the superior vena cava and the atrium. The number 2 represents the auricle, and the optimal position would be approximately 1 cm. higher. Note that this point is rather low on the cardiac silhouette. *B*, Oblique view, emphasizing the rather low position of the caval-atrial junction. *C*, Postoperative roentgenogram demonstrating actual placement of the caval catheter.



A B

Fig. 128. *A*, Preoperative appearance of a patient who suffered fulminating hydrocephalus following complete removal of a large cerebellar astrocytoma. *B*, Postoperative appearance after use of the ventriculo-caval shunt.

A

B

Fig. 129. *A*, Newborn infant with large occipital encephalocele. Neurologic evaluation suggested a satisfactory developmental potential. *B*, The same patient after operation, with the ventriculo-caval shunt in place. The child's development has been normal thus far (age 5 months).

Fig. 130. Typical appearance of a decompressed anterior fontanel after a ventriculo-vascular shunt.

Fig. 131. Two children originally rejected for operation because of large heads and thin brains. Ventriculo-caval shunts were carried out because of preservation of affect.

The following simple developments have reduced the revision rate from 37 to 22 per cent in a 3-year period: (1) the development of more precise and dependable pressure regulation in the valve apparatus; (2) the development of the molded silicone right-angle ventricular catheter for the patient with relatively thin brain substance, and the development of a closed-tip straight catheter with multiple small openings at the tip for the patient with thicker brain tissue; (3) the development both of x-ray and image intensifier techniques for precise placement of the venous catheter (Fig. 127).

In summary, we at The Children's Hospital of Philadelphia, in common with many of our pediatric colleagues, are encouraged by the many excellent results we see following operation in hydrocephalus, and at present see no justification for pessimism in the therapeutic outlook (Figs. 128 through 131).

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